

APPLIED KINESIOLOGY

VOLUME II

Head, Neck, and Jaw
Pain and Dysfunction —
The Stomatognathic System

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David L. Waters, D.C.



APPLIED KINESIOLOGY

VOLUME II

**Head, Neck, and Jaw
Pain and Dysfunction —
The Stomatognathic System**

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Table of Contents

Acknowledgements	ix
Preface	xi
Preface	xiii
Introduction	xv

Section I

Cranial-Sacral Primary Respiratory Mechanism

Chapter 1 — Introduction	3
Chapter 2 — Introduction to Cranial Function	9
Skull Motion	10
Objective Measurement of Motion	17
Role of the Cranium in Health and Dysfunction ...	20
Cranial Nerves	20
Nerve Receptors Within Sutures	20
Cerebrospinal Fluid	20
Blood Pressure	22
Circulation to the Brain	22
Endocrine Disturbance	22
Visual Disturbances	23
Auditory	23
Neurologic Disorganization	23
Temporomandibular Joint Dysfunction	23
Holographic Concept	23
Etiology of Cranial Faults	24
Trauma	24
Structural Imbalance	26
Cranial Muscles	27
Habit Patterns	27
Chemical	27
Chapter 3 — Cranial Osteology	29
Introduction to Cranial Anatomy and Physiology ..	29
General Landmarks	30
Cranial Primary Respiration	35
Skull Syndesmology — Introduction	39
Types of Sutures in the Skull	40
Limited Motion of Joints	42
Sphenoid Bone	43
Occipital Bone	50
Temporal Bone	53
Frontal Bone	61
Parietal Bone	64
Maxillary Bone	65
Palatine Bone	67
Zygomatic Bone	69
Ethmoid Bone	70
Vomer Bone	71
Nasal Bone	72
Inferior Nasal Concha	72
Lacrimal Bone	73
Meninges	74
Integration of Cranial Movement	76
Embryology	77
Correlation of Skull Movement	79
Skull Movement	79
Cranial Vault	79

Vault Bones Ossifying from Membrane	83
Facial Bone Movement	84
Evaluating Skull Motion	87

Chapter 4 — Skull Muscles	89
Muscles Involved with the Stomatognathic System	89
Muscles and Fascia of the Cranium and Face ...	89
Cranial Muscles and Fascia	90
Subcutaneous and Deep Fascia	90
Occipitofrontalis (Epicranius)	91
Temporoparietalis	92
Extrinsic Muscles of the Ear	93
Auricularis Anterior	93
Auricularis Superior	93
Auricularis Posterior	93
Muscles of the Eyelids	94
Orbicularis Oculi	94
Corrugator Supercilii	95
Levator Palpebrae Superioris	95
Muscles of the Nose	96
Procerus	96
Compressor Naris	97
Depressor Septi	97
Dilator Naris	97
Muscles of the Mouth	98
Levator Labii Superioris	98
Levator Labii Superioris Alaeque Nasi	98
Levator Anguli Oris	98
Zygomaticus Minor	99
Zygomaticus Major	100
Risorius	100
Depressor Labii Inferioris	100
Depressor Anguli Oris	101
Mentalis	101
Buccinator	101
Orbicularis Oris	102
Other Muscles Influencing the Cranium	103
Muscles of Mastication	103
Hyoid Muscles	103
Postural Muscles	103

Chapter 5 — Cranial Nerves	105
Cranial Nerve Introduction	105
Format of Cranial Nerve Presentation	106
Anatomy and Distribution	107
Possible Entrapment	107
Examination	107
I — Olfactory Nerve	108
II — Optic Nerve	109
III — Oculomotor Nerve	111
IV — Trochlear Nerve	112
VI — Abducent Nerve	113
Examination of Cranial Nerves III, IV, and VI	114
Cardinal Positions of Gaze	114
V — Trigeminal Nerve	116
Ophthalmic Nerve	116
Maxillary Nerve	116
Mandibular Nerve	118

VII — Facial Nerve	122
VIII — Acoustic Nerve (Vestibulocochlear Nerve)	125
Cochlear Nerve	125
Vestibular Nerve	125
IX — Glossopharyngeal Nerve	128
X — Vagus Nerve	130
XI — Spinal Accessory Nerve	132
Cranial Part	132
Spinal Part	132
XII — Hypoglossal Nerve	135
Differentiation of the Etiology of Cranial Nerve Dysfunction	136

Chapter 6 — Examination and Treatment of Cranial Faults

Introduction	139
Diaphragmatic and Cranial Respiratory Correla- tion	139
Body Language of Cranial Faults	142
Health Problems	142
History	143
Asymmetry	144
Remote Problems	148
Types of Examination	148
Visual Observation	148
Palpation	149
Applied Kinesiology	150
Format of Cranial Fault Examination and Correc- tion	151
Mechanics of the Fault	152
Mechanics of Correction	152
Therapy Localization of Cranial Faults	152
Challenge	153
Muscle Correlation	155
Pain Location	155
Conditions Correlating	155
Correction	155
Flexion-Extension Group of Cranial Faults	157
Inspiration Assist Cranial Fault	157
Expiration Assist Cranial Fault	162
Sphenobasilar Inspiration Assist Cranial Fault ..	165
Sphenobasilar Expiration Assist Cranial Fault ..	167
Combined Inspiration and Expiration Assist Cranial Faults	170
Glabella Cranial Fault	171
Rotational Cranial Faults	174
Temporal Bulge Cranial Fault	174
Parietal Descent Cranial Fault	177
Internal Frontal Cranial Fault	180
External Frontal Cranial Fault	184
Universal Cranial Fault	187
Sutural Cranial Faults	190
Sagittal Suture Cranial Fault	190
Squamosal Suture Cranial Fault	191
Lambdoidal Suture Cranial Fault	193
Zygomatic Suture Cranial Fault	194

Chapter 7 — Examination and Treatment of Pelvic Dysfunction

Introduction	197
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Pelvic Anatomy	198
Sacrum	198
Coccyx	199
Innominate Bone	199
Sacroiliac Articulation	200
Primary Respiratory Motion of the Pelvis	201
Sacral Respiratory Movement	201
Coccyx	203
Innominate Movement	204
Pelvic Primary Respiratory Examination and Treat- ment	205
Sacral Inspiration Assist	205
Sacral Expiration Assist	207
Coccyx	210
Category I Pelvic Fault	211
Category I Abdominal Muscle Weakness Chart	212
Cranial-Sacral Correlation to Cloacal Synchroni- zation Technique	214
Sacral Wobble	217
Atlanto-Occipital Countertorque	219
Recurrent Cranial and Pelvic Faults	220

Section II Stomatognathic Area

Chapter 8 — Stomatognathic Area Introduc- tion

Terminology	225
Teeth	226
Surfaces of Teeth	228
General Terms	229
Orthodontic Nomenclature	230
Angle Classifications	230
The Stomatognathic System's Role in General Health Problems	232
Triad of Health	238
Summary	240

Chapter 9 — Anatomy of Muscles of Mastica- tion, Tongue, and Teeth

Introduction	243
Masticatory Muscles	244
Temporalis	244
Masseter	247
Internal Pterygoid (Medial Pterygoid)	248
Mandibular Sling	250
Buccinator	251
External Pterygoid (Lateral Pterygoid)	252
Organization of Muscles in Mandibular Movement	254
Resting Activity	254
Normal Jaw Aperture	255
Hyoid Group	256
Tongue Anatomy	256
Gross Appearance	256
Muscles of the Tongue	257
Extrinsic Muscles	257
Intrinsic Muscles	258

Periodontal Ligament	259
Function	260
Neuroanatomic Basis of Mandibular Movement	261
Prodigious Nerve Supply	263
Challenge and Manipulation of Teeth	265

Chapter 10 — Cranial and Stomatognathic

Area Interdependence	269
Mandibular Influence on Cranial Function	270
Normal Masticatory Action	271
Swallowing	273
Head-to-Shoulder Girdle Musculature	275
Postural Muscles	275
Muscles of Mastication	278
Hyoid Muscles	278
Muscles of Head-on-Shoulder Girdle	
Correlation	280
Development of Neurologic Disorganization	282
Model of Primary and Secondary Conditions	283
Disorganization within the Stomatognathic	
System	285
Iatrogenic Conditions in the Stomatognathic	
System	286

Chapter 11 — Temporomandibular Joint

General Description	291
Anatomy	293
Mandible	293
Temporomandibular Articulation	294
Mandibular Positions	301
Rest Position	302
Freeway Space	305
Centric Relation and Centric Occlusion	306
Form vs. Function	310
Disturbed Centric Relation	310
Cranial Fault	311
Muscular Imbalance	311
TMJ and Mandibular Movement	312
Rotation and Translation	313
Methods of Measuring Mandibular and	
TMJ Motion	319
Mechanical Methods	319
X-ray and Photographic	319
Mandibular Kinesiograph	319
Class of Lever	325
TMJ Internal Derangement	328
Disc Dysfunction	328
Conservative Treatment	333
Surgery	337
Trismus	337
Dislocation	338

Chapter 12 — Occlusion

Optimum Occlusion	344
Etiology of Malocclusion	345
Occlusion and Stability of Teeth	347
Remodeling	347
Periodontal Disease	348
Cranial Influence on Structure	348
Muscular Balance	350

Vertical Dimension	356
Iatrogenic Causes of Malocclusion	358
Considerations Prior to Equilibration	359
Screening Examination for Prematurities	360
Phonetic Occlusal Evaluation	360
Palpation	361
Wax Bite	361
Marking Methods	361
Photocclusion	362
Mandibular Kinesiograph	363
Bite Plane-Plate-Splint	365
Bite Planes for Neuromuscular Influence	365
Bite Plane to Supplement Therapy	367
Establishing Vertical Dimension by Use of	
Bite Planes	370
Bite Planes for Protection of the Teeth	374
Equilibration	375

Chapter 13 — Hyoid Muscles and Function

Introduction	383
Hyoid Bone Anatomy	383
Muscles of the Hyoid	386
Suprahyoid Muscles	386
Infrahyoid Muscles	390
Hyoid Muscular Balance	394
Body Language	395
Hyoid Neuromuscular Balance Examination	
and Treatment	395

Chapter 14 — AK Examination and Treatment of the TMJ and Muscles of Mastication

Introduction	401
Examination	402
History and Consultation	402
Observation	406
Palpation	406
Differential Diagnosis	409
Intracapsular TMJ Pathology	410
Extracapsular Disturbance Referred	
to the TMJ	412
Therapeutic Methods	414
Cranial-Sacral Primary Respiratory System	414
Proprioceptors	415
Spray and Stretch	416
Fascial Release	420
Reflexes and Meridians	420
Applied Kinesiology TMJ Evaluation	421
No Therapy Localization to the Temporo-	
mandibular Joint	422
Centric Occlusion	422
Jaw Movement Without TL	423
Jaw Forced Open Without TL	423
Therapy Localization to the Temporo-	
mandibular Joint	424
TL to the TMJ — No Movement	425
Isometric Contraction With Therapy	
Localization	425
Jaw Movement in Sagittal Plane With TL	425

Jaw Movement in Transverse Plane	
With TL	431
Sequential Mandibular Movements	
With TL	433
Mandibular Movement Plus Remote	
Factors	434
Chapter 15 — Myofunctional Therapy	439
Introduction	439
Muscle Envelope	442
Etiology of Orofacial Muscle Imbalance	447
Deviant Swallowing Patterns	447
Cause of Tongue Thrust	447
Examination	452
Habit Pattern Correction	458
Myofunctional Therapy Exercises	460
Lip Exercises	461
Training Proper Tongue Function	469
Training a Permanent Habit	470
Final Considerations	470

Section III

Organization Between Systems

Chapter 16 — Examination of the Integrated Stomatognathic System	477
Body Language	478
Cranial Faults	479
Dental Conditions	480
Neurologic Disorganization	481
Postural Deviation	481

General Symptoms	481
Trauma	481
Sequential Steps of Examination and	
Correction	482
Muscle Proprioceptors	484
Muscle Stretch Reaction	484
Cranial Correction	487
Stomatognathic Area	480
Hyoid	488
Integration of the Stomatognathic System	488
Troubleshooting the Stomatognathic System	489

Chapter 17 — Stomatognathic System

Organization to Total Body	495
Modular Organization	495
PRYT	496
Eyes Into Distortion	500
Weight Bearing	502
Gait Mechanism	505

Chapter 18 — Relation of the Stomatognathic System With Various Health Problems

Conditions	511
Locating the Cause of Neurologic	
Disorganization	515
Dynamic Use of K27 as an Indicator	
for Switching	517
Correction of Switching in the Clear	518
Finding and Correcting a Hidden Positive K27 ..	519

Index	525
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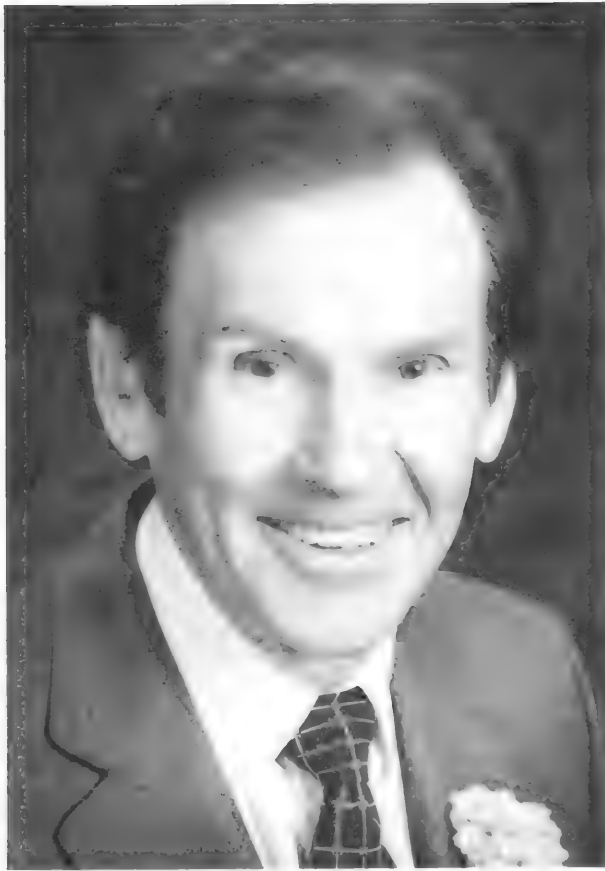
Developing rough manuscript to its final form requires knowledge, persistence, and dedication. My secretary, Carol Ann Hupp, not only provides this level of expertise, but does it in a pleasant and warm manner, even under the stress of deadlines. Her ability to structure the written word has improved the syntax of this text.

The majority of the material in this text is a compilation of the work of others. My appreciation — and so should that of all others in the healing arts — goes to the creative thinkers, research scientists, and authors who have presented material before. We have all called on their work, too often failing to remember that what can be accomplished today is directly related to their efforts in sharing. As knowledge grows, the vast amount of background information comes better into perspective.

Along with the standard anatomy, physiology, and chiropractic body of knowledge, I have throughout this text relied on information from the *Collected Papers of the Members of the International College of Applied Kinesiology*. Because of the members' efforts to share with their fellow physicians, we all benefit.

Finally, this material is dedicated to you, the practicing physician, with the sincere hope that it will aid many thousands of individuals in obtaining health through natural methods.

Preface



It is difficult to imagine that an efficient prophylactic or therapeutic regime against any deformity or disability can be instituted by anyone not acquainted with the normal kinetics of the human body. Further, a knowledge of the conditions under which the body maintains equilibrium is the best way to assess pathologic situations. This thought was put forth by that famous orthopedist, Dr. Arthur Steindler, in his book *Kinesiology of the Human Body*. This same idea has been expanded here by Dr. David Walther.

Another simple fact is of fundamental significance. In order for joints to be stable, they must be maintained by muscular forces. This is because ligaments by their design are only checkers and restrainers of sudden impact. Around each normal joint there must be an equilibrium of forces. Forces in one direction must cancel out the forces in the opposite direction, so that they add up to zero. This is a fundamental principle of applied mechanics. If such is not the case, displacements occur. If not immediate, they take place subtly over long periods of time and produce a variety of clinical problems.

Homeostasis pertains to the maintenance of the

body's skeletal parts or of the individual tissues and glands. Therefore, not only physical equilibrium is brought into consideration but also certain factors of body chemistry are significant. The contemporary position a clinician takes is concerned with total equilibrium. Parts may be affected from conditions far removed anatomically from the parts involved. Noxious elements may cause instability in a variety of tissues at distant sites. It is precisely this chain of reasoning that is implicit in Dr. Walther's presentation. He has first established an in depth view of anatomy. He then proposes a logic and rationale of the tissues. From a recourse to the literature of the various theories proposed by others, he has selected a most appropriate idea, and those in agreement with his own discipline, experience, and observations.

From a viewpoint of totality in the body, he has explained his beliefs and offers the necessary factors to help integrate the various disciplines in the health sciences. It is becoming more obvious that this bilateral approach is gaining in public acceptance. It is further quite evident that the drug approach to pain control and the use of chemicals to create an artificial sense of well-being is beginning to run its course. Every indication points to the need to return to the forces of nature and to the consideration of factors contributing to normal equilibrium and homeostasis. This is the basis for clinical practice as Dr. Walther perceives it.

The original meaning of physician was "one who works with the ways of nature." Historically, the first surge of health care originally worked with the physical world. This preceded the "medical" doctor. To this day, many chemicals employed for clinical control were derived from herbs or started from some of nature's old remedies. Dr. Walther has now put one more turn on the clock of time with regard to health care using an approach of physical welfare as a basis for bodily problems. He has brought us that much closer to our goal and constant knowledge and skill in dealing with locomotor disorders. He has made an outstanding contribution to our quest for the ability to maintain the physical, mental, and social well-being of our patients. Possibly and most significantly, he has made a great step in communication with the allied disciplines in chiropractic, osteopathy, dentistry, medicine, and physical therapy. To do this is to be congratulated. The student should find great inspiration throughout the course of these texts.

*Robert M. Ricketts, D.D.S., M.S.
Pacific Palisades, California*

Preface



Rapid growth continues in the body of knowledge known as applied kinesiology. This growth can be attributed primarily to the contribution this system is making in evaluating functional disturbances. Much of the effort in the healing arts has been directed toward the classification and treatment of pathology. Many pathological conditions were at one time functional disturbances that went uncorrected. Certainly applied kinesiology contributes to the diagnosis and understanding of pathological conditions, but its primary value is in understanding and diagnosing functional disturbances.

The use of applied kinesiology must be placed into proper perspective. It is a system which gives added dimension to diagnosis, and therefore should be combined with standard physical diagnosis, laboratory, x-ray, history, and the other special examination procedures of any physician using applied kinesiology as an adjunct to diagnosis. AK examination should enhance standard diagnosis, not replace it.

This series of texts is intended for the use of physicians of various disciplines such as chiropractic,

dentistry, medicine, osteopathy, podiatry, and optometry. These individuals are licensed to be primary health care providers, and are thus qualified to make initial diagnosis, provide treatment, and make referrals when necessary. Some who are not qualified by law or education have taken a few applied kinesiology procedures and used them in treatments which may or may not be correct when examination is placed into total perspective with the procedures used in the various disciplines of the healing arts. In most cases when manual muscle testing is used in this limited way, the person making the examination has only a small repertoire of testing procedures. There are even instances where lay people are selling nutrition by attempting to evaluate it with limited manual muscle testing. This limited and inappropriate use of manual muscle testing for evaluation of health and a person's nutritional needs is strongly condemned by the International College of Applied Kinesiology. It must be recognized that applied kinesiology is a discipline which requires extensive study, and it must be combined with a physician's expertise learned in graduate study, post-graduate study, and from experience.

When put into proper perspective, applied kinesiology adds a new dimension to diagnosis. It is valuable primarily in functional conditions; it helps the physician understand symptomatic complexes which are not pathological in nature, as well as why pathology has developed.

This text is the second in a series of five volumes. Volume I presents most of the basic procedures used in applied kinesiology, as well as the application of manual muscle testing. Volumes III, IV, and V will be published sequentially as they are completed. Volume III primarily covers the meridian system and other electromagnetic factors of body function. Volume IV is on orthopedic conditions, and Volume V on systemic conditions (including metabolic involvement, the endocrine system, digestive function, etc.).

Few of the ideas and procedures in these volumes are original, but they have been used in a large clinical practice. A "thank you" goes to George Goodheart for his many original concepts and continued efforts in teaching others. When chiropractors over the years have expressed a desire that he keep his knowledge within the chiropractic profession, he has repeatedly said, "You can only keep what you give away." This is so true.

*David S. Walther, D.C.
Diplomate, ICAK*

Introduction



The privilege and opportunity of writing an introduction to this second in a series of carefully documented and edited books is interesting since it allows me to quote myself. "The organizational genius of David Walther has produced a growing flood of physicians of all disciplines well-trained in applied kinesiological principles." It also offers me the opportunity to quote my good friend, Harold Gelb, D.M.D., who is the clinical professor at the College of Medicine and Dentistry in New Jersey and the director of the Temporomandibular Joint Clinic of the New York Eye and Ear Infirmary, New York. He and other authors in his text, *The Clinical Management of Head, Neck and TMJ Pain and Dysfunction*, which is a multi-disciplinary approach to the diagnosis and treatment of temporomandibular joint and associated problems, have indicated that applied kinesiology is concerned with the dynamics of the smooth and striated musculature and the impact of these functions on structural entities, healing processes, and disease resistance. In particular, applied kinesiology focuses on the identification and the correction of proprioceptive dysfunctions of ligaments and of muscle spindle cells and Golgi tendons. Finally, applied kinesiology is con-

cerned with the vascular, lymphatic, and other systems supporting proper muscle dynamics, as well as the nutritional requirements necessary for these support systems and the muscles themselves.

Dr. Clyde H. Schuyler, D.D.S., formerly the professor of prosthetic dentistry at New York University's College of Dentistry, said in his foreword to Nathan Allen Shore's book, *Temporomandibular Joint Dysfunction and Occlusal Equilibration*, "The correction of occlusal disharmonies of the natural dentition can be accomplished effectively by selective and judicious spot grinding. If it is done injudiciously without a careful preliminary study of the interfering occlusal contacts, it may not only fail to relieve the trauma, but may in turn contribute to additional oral discomfort." The need to create a favorable postural relation between the maxillae and the mandible allows the creation of a functional harmony of both upper and lower teeth, which then allows the distribution of a balanced relationship of occlusal stress, both in the eccentric and centric relationships of the mandible. There are many factors that can influence this harmonious potential relationship, but a primary and simple observation began early in 1964 with the concepts of applied kinesiology.

The basic and continuing concept of applied kinesiology relates itself to the pattern that muscle weakness is primary in most muscle spasm and is causal, both in its diagnosis and in therapy. Most muscle spasm occurs because of related muscle weakness. The three-sided triangularity of man — structural, chemical, and psychological — with the five-factored IVF circle superimposed on the triangle, continues to be a means of understanding the problems of both biped man with his quadrupedal nervous system and the quadrupedal field of veterinary science. Continuing lectures at leading dental schools and the convening of multi-disciplinary symposia have allowed many dedicated health professionals to come closer to a holistic concept which unifies rather than divides the healing arts. Lectures and demonstrations continue to be organized by interested members of all disciplines in many countries of the world. Norway, England, France, Japan, and Australia continue to be visited and re-visited with updated and new material presented by many traveling AK diplomats. Scientific documentation in all the healing arts is accumulating in both the private professional sector and at established colleges and universities. Applied kinesiology, although multi-disciplinary in scope, was and is chiropractic in origin and function, and thus has contributed to both the inter- and intraprofessional dialogue which has

benefitted patients, doctors, and the scientific community.

"Numquam mandacium dictio corporea dicit."

This Latin phrase in a basic translation cites the obvious pattern that body language never lies. The opportunity of understanding body language is enhanced by the therapist's opportunity to use muscles as indicators for body language. The original method of testing muscles and determining their function was based upon the observations of Kendall, Kendall, and Wadsworth; this approach remains the prime diagnostic art and method. Once muscle weakness, or in the rare instance muscle spasm, has been diagnosed, a variety of therapeutic options are available, which are too numerous to cite here. Diagnosing the need, supplying the need, and observing the result are the primary efforts of the physician using applied kinesiology. The opportunity to use the body as an instrument of laboratory analysis is unparalleled and unequalled in modern therapeutics because the response of the body is unerring. If one approaches a problem correctly, making the proper and adequate diagnosis and treatment which are supported by standard methods of observation — as well as by applied kinesiology techniques — the response is adequate and satisfactory, both to the doctor and to the patient.

The body heals itself by primary intent. It heals itself in a sure, sensible, practical, reasonable, and observable manner. The healer within can be approached from without. Man does possess a potential for recovery through the physiological homeostatic innate intelligence of the human structure. This recovery potential with which he has been

naturally endowed and which is his natural birthright merely waits for the trained hand, the knowing mind, and the caring heart of the trained individual to bring it to physical manifestation, allowing health to come forth; this is man's natural heritage. This benefits man, both individually and collectively, and also the physician or therapist who has rendered the service. It allows the same force which created the body to heal the body and allow it to operate unimpeded. These benefits can be performed and derived with knowledge, with the application of physiological facts, and with predictable certainty. It should be done, it can be done, and these texts offer a means and a measure of how it can be done. My continued thanks to Dr. Walther and his staff for his performance in advancing these principles, and as always my best wishes are extended to all who read this manual and the succeeding volumes which are planned for the future.

Dr. Gelb's early quotation has come to active fruition: "We believe that medical and dental specialists will use applied kinesiological testing to make more accurate diagnosis and provide better treatment. But that is a futuristic thought." The future begins to be now. David Walther, by his careful application of research and scientific principles, has allowed sound and sensible knowledge of the musculoskeletal system, especially in the stomatognathic area, to come to early fruition. My thanks again to him and his staff.

*George J. Goodheart, Jr., D.C., FICC
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Section I

**Cranial-Sacral
Primary Respiratory Mechanism**

Chapter 1

Introduction

Applied kinesiology considers the body as a total, integrated whole. One of the great strengths of this approach to examination is the ability to observe and evaluate integrated body activities. Although it is important to consider the entire body, this text is limited to discussing the cranial-sacral primary respiratory mechanism and the stomatognathic system. The evaluation and treatment of this system influences a wide range of conditions.¹⁷ The approach, however, is not a panacea; it should be integrated with the rest of the material in applied kinesiology and standard physical diagnosis.

The term “stomatognathic” usually refers to the mouth and jaw. Here we are looking at a more comprehensive unit and refer to the stomatognathic system. Shore²⁰ states that the stomatognathic system has components of “. . . the bones of the skull, the mandible, the hyoid, the clavicle and the sternum; the muscles and the ligaments; the dento-alveolar and the temporomandibular joints; the vascular, the lymphatic and the nerve supply systems; and the soft tissues of the head; the teeth.” To obtain a more comprehensive approach to the system, this writer feels it is necessary to include the spinal column and the pelvis because of the closely integrated activity that will be presented throughout the text. As support for including so much structure under the term “stomatognathic system,” Shore goes on to explain that an organ — such as the heart or liver — can be dissected anatomically, but a system such as the stomatognathic must be studied as an integrated, physiologically functioning whole. We agree; it is necessary to study the interactions of the structures comprising the system, rather than studying individual tissues and structures as single entities. Because of the integration of the spinal column and the pelvis with the cranial primary respiratory mechanism, it is impossible to leave them out of this discussion.

The integration of the stomatognathic system with the spinal column is related by Fonder,⁸ who

points out that some of the muscles of the head, neck, and shoulder girdle are involved in the masticatory system. Specifically, the hyoid muscles serve as a connection between the chewing mechanism and the shoulder girdle. The influence of the muscle proprioceptors on this area from disturbed mastication can produce improper information regarding the head-on-body relationship. There is further integration by way of the extensive fascial investment of the cervical region connecting with the masticatory mechanism. Berkman¹ has shown a consistency of the short or reactive leg length and the laterality of the atlas to the maloccluded side of the temporomandibular joint. Fonder⁹ has demonstrated the influence of the stomatognathic system on body balance by changing the occlusion and mandible position. Before and after x-rays of the spinal column show significant changes in structural balance. Changes throughout the body, often seen on a clinical basis after making changes in the masticatory system, show how the structures within the stomatognathic system are integrated with each other, and the system is related to total body activity. (See figures 1—1 through 1—6, pages 4-5.)

Although it is necessary for all physicians to be knowledgeable about the system and how to examine its function, there is no single profession which can provide all the services necessary to correct the various types of dysfunction found in this highly complex area. A specialist in one area of the healing arts may observe dysfunction in one of the structures and initiate treatment; however, if there are unnoticed areas which function improperly, the efforts of the therapy will be unrewarding or will fail to return the individual to the highest plateau of health. Apparent corrections may be obtained, only to be lost because of the interrelating activity of some uncorrected factor.

A dentist highly trained in the fine art and science of equilibration may direct his efforts toward improving an occlusion to relieve temporomandibular joint pain, headaches, or dental trauma from mastication;

Introduction

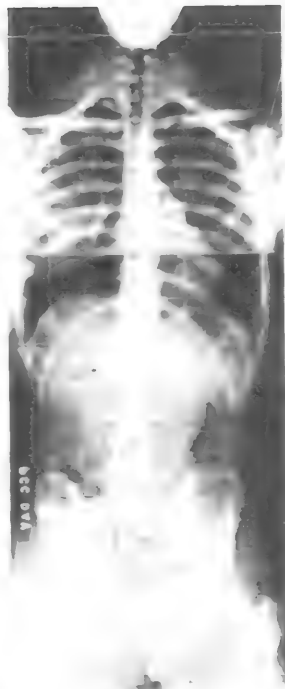
Illustrations from *Basal Facts* (Fall 1976) with permission of
Aelred C. Fonder, B.A., D.D.S., FRSH.

Case #1



Before treatment

1—1. Anteroposterior view



After treatment



Before treatment

1—2. Lateral view



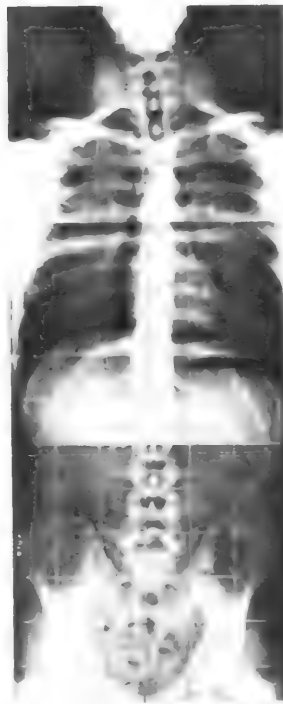
After treatment

Case #2



Before treatment

1—3. Anteroposterior view



After treatment



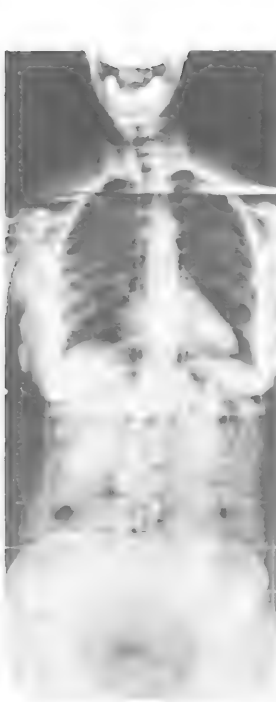
Before treatment

1—4. Lateral view

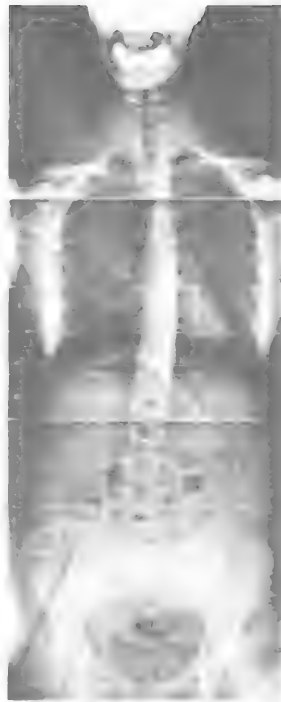


After treatment

Case #3



Before treatment



After treatment

1—5. Anteroposterior view



Before treatment



After treatment

1—6. Lateral view

yet the patient's structure, being unstable, continually shifts and requires re-equilibration. This may be due to cranial dysfunction continually changing the occlusion or muscle imbalance. On the other hand, a physician knowledgeable in cranial function may make corrections, only to find the problem returning because there is an occlusal problem. Every time the patient chews, imbalanced mechanical pressures are forced on the cranium by the masticatory muscles, thus re-creating the cranial faults. The occlusion may have been correct prior to the cranial treatment, but because of the change in the cranial bones, there is now malocclusion. In order for cranial corrections to be maintained, adjustment of the occlusion — to match the new position of the cranium — may be necessary. This often requires the dentist to be familiar with evaluating the muscles of mastication for balance, as well as evaluating the skull for cranial faults. There has been some material written about AK muscle balancing techniques in dental literature,^{4, 5, 6, 7, 12} but much more effort is needed. This is particularly true in light of some of the controversy which has developed as a result of the holistic approach being used in dentistry.^{13, 14, 16, 18, 19}

It is especially important that the body's integrated activity be recognized because structural dysfunction throughout the body can be related to the stomatognathic system. This may include dysfunction of the feet or pelvis which creates improper nerve impulses through the proprioceptive system, causing muscles of the neck and head to be facilitated or inhibited at inappropriate times for the activity being performed. Such muscular imbalance can create a cranial fault, which in turn may change the patient's occlusion. The primary complaint of the individual may be about dental health; the basic underlying cause is not recognized.

The same illustration can be applied to many other facets of health. Disturbance in the cranial-sacral primary respiratory mechanism can influence vision, manifested either in a decrease of visual acuity or the efficiency of the eyes working well together. The optometrist or ophthalmologist may prescribe a refraction for the visual acuity problem, or prisms for diplopia. This will symptomatically improve the situation; however, it leaves the basic underlying cause uncorrected. On the other hand, a doctor knowledgeable about cranial primary respiratory

Introduction

function can return normal control to the extraocular muscles, but rehabilitation exercises in a visual training program may be needed.

Within the stomatognathic system there is a close integration of nerve function which is important to total body organization. Again, there is certainly a two-way street between various professions in dealing with this problem. The proprioceptors of equilibrium — such as the labyrinthine and visual righting reflexes, and neck righting reflexes — all work together and potentially can be adversely influenced by various mechanisms. The dentist improving cranial primary respiratory function by way of occlusal corrections may improve activity of the labyrinthine and visual righting reflexes; yet he fails to obtain optimum function because there is still an involvement of the upper cervical region influencing the neck righting reflexes. This condition may require a chiropractor to make corrections in the cervical spine and possibly other areas of the spine and in the pelvis.

Examples of the many ramifications of health problems caused by dysfunction in this system could go on and on. It is sufficient here to emphasize the necessity of different specialty health professionals being knowledgeable about the mechanism so that optimum examination is done for the patient's best benefit. Appropriate referrals and communication between specialists are of great importance.

The approaches discussed here for the examination and treatment of the stomatognathic system must be integrated with other applied kinesiology procedures presented in Volume I of this series. For example, to evaluate the neck righting reflexes and their organization with the visual righting and labyrinthine reflexes, it is necessary to evaluate the organization of the body's modules and cloacal synchronization. This is presented in Volume I under "PRY Technique" and "Cloacal Synchronization." There is a further discussion of PRYT Technique in Chapter 17 of this volume.

Within the stomatognathic system there are mechanisms which could be considered systems in themselves, or sub-systems. The cranial-sacral primary respiratory mechanism may appear to be an autonomous system; however, it is significantly influenced by the strong muscles of mastication and by muscles such as the sternocleidomastoid, upper trapezius, etc., which are head-balancing muscles. On the other hand, cranial-sacral dysfunction can cause the TMJ, associated muscles, and structures to become involved. The muscles of the hyoid correlate with swallowing, phonation, and mastication; they also appear to have some function relating to body balancing and other neurologic organization

through their muscle proprioceptors.

Although the stomatognathic system as described here has a major influence on body function, it has been the subject of very little comprehensive discussion in the literature. There is considerable reference to parts of the stomatognathic system. Shore,²⁰ in describing the components of the stomatognathic system, gives an excellent presentation of the dental side of examination and treatment. Gelb edited a book, *Clinical Management of Head, Neck and TMJ Pain and Dysfunction*,¹¹ which gives a multidisciplinary approach to diagnosis and treatment, including discussions of various dental procedures. Also offered are brief descriptions of cranial osteopathy and applied kinesiology, biofeedback, myofunctional therapy, and several other therapeutic approaches. Magoun,¹⁵ a leader in cranial osteopathy, reviewed the chapters of Gelb's book and pointed out how important the cranial-sacral primary respiratory system is in correcting craniocervical pain. Char has been active in teaching courses on holistic dentistry and has published two texts^{2,3} which cover numerous approaches to the stomatognathic system, including nutrition, myofunctional therapy, cranial osteopathy, and a modification of applied kinesiology, among other procedures. Eversaul⁶ presents applications of applied kinesiology to dentistry, but he fails to discuss the cranial primary respiratory mechanism. Garliner¹⁰ pays particular attention to the facial and masticatory muscles in his myofunctional therapy. Although not correlated specifically to cranial respiratory dysfunction, these procedures show the importance of muscular function associated with the cranial mechanism. Fonder⁹ shows the influence of the stomatognathic system on total body function. He particularly discusses the "dental distress syndrome"⁸ and its influence on health. As Selye⁹ states in his introduction to Fonder's text, "I fully support his plea to dentists to realize that they are 'treating persons who are united wholes, not simply a complex of dental and periodontal tissues.' I can only hope that others in the field take up the challenge and investigate in their specialty those areas that hold great promise, not only to the patient and the dentist, but also to the practicing physician."

The main point is that many structures and systems can be involved in craniocervical pain or dysfunction. The important factor is the ability to evaluate all factors to find the total involvement and the basic underlying cause(s). Correction may require referral to another discipline.

The goal of this text is to present the approaches used in applied kinesiology to evaluate and treat the stomatognathic system. Regardless of one's profession, it is necessary to learn how all areas of the body

interrelate. There is no professional group which has the expertise necessary to effectively treat all dysfunction with which it comes in contact. As professionals in the various disciplines work with this system, greater knowledge of the interrelationships will be gained. With continued interdisciplinary exchange, the approaches will be expanded for the benefit of all concerned.

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Chapter 2

Introduction to Cranial Function

The cranium has inherent motion that is necessary for life. The bones of the skull move in a cyclic synchronism of 10 to 14 cycles per minute. This activity has been termed "the primary respiratory mechanism."⁶²

Along with the cranial motion is a synchronous motion of the sacrum and pelvis. This correlated activity occurs because of the mechanical communication between the skull and pelvis via the dural mechanism covering the brain and spinal cord and attaching to the sacrum.^{12, 13, 15, 46, 62} There probably is also influence from the extensive fascial connections and muscle activity. The abdominal muscles are especially active in pelvic respiratory motion. The cyclic motion of the cranial bones and the pelvis is an independent motion which sometimes parallels breathing and has other correlations to the rhythmic motions of body function.

Cranial bone movement has been a controversial subject in the healing arts. Members of the osteopathic and chiropractic professions have been especially vocal about this subject. Few colleges in the healing arts emphasize — or even recognize — skull motion as an important aspect in physiology. Objective studies in more recent years support the findings of the early pioneers in this field. Clinicians actively applying cranial therapeutics have expanded on the application with clinical results as indication of the validity of their systems.⁴²

It appears that investigation into the possibility of cranial motion began at approximately the same time in both the chiropractic and osteopathic professions. Sutherland's interest in cranial bone motion developed in 1899 while he was an osteopathic student at The American School of Osteopathy in Kirksville, Missouri (Note: now the College of Osteopathy and Surgery). During his practice of osteopathy, Sutherland continued his thoughts about the possibility of cranial motion. With ingenious devices, he applied static pressure to his own head to observe any changes that might take place in his body function.

After years of cranial study, he first presented ideas about the cranium to his profession in 1929,^{61, 62} and in 1939 he published his text, *The Cranial Bowl*, which delineated the movement of the skull. His early description of skull motion appears to be accurate, with some of the motions more recently having been measured with electrical force transducers.^{20, 51, 62} It was Sutherland who developed most of the terminology used today in reference to cranial therapy.⁶⁸

Nephi Cottam, a chiropractor, began considering the possibility of skull motion in 1895 at the early age of 12. Upon observing the skull of an animal drying in the hot desert sun, he noticed the bones of the skull separating from one another. He later related to his son, Calvin Cottam, that he believed this was his first observation of motion of the skull bones.⁸ Cottam also began teaching the professions in 1929, and published a small book on the *Story of Craniopathy* in 1936.¹⁰ In 1937 Cottam made a tour of osteopathic colleges, demonstrating cranial technique in the company of John C. Burnett, D.C.⁸

From these beginnings, many have researched cranial function and expanded on the early pioneers' thoughts. Today cranial therapeutics are primarily practiced by three groups. (1) There are osteopaths — and some dentists — who primarily follow Sutherland's teachings. (2) Chiropractors use a combination of many therapeutic approaches. It appears the most organized group in chiropractic — outside of applied kinesiology — is that which uses the sacro occipital technique developed by Major Bertrand DeJarnette.^{12, 14} Calvin Cottam and Reid Rasmussen teach the Cottam approach to cranial adjusting.⁹ James R. Alberts, D.C., Leo Spears, D.C., and D. J. Metsinger, D.C., are others who pioneered cranial therapy in chiropractic. (3) Applied kinesiologists have adapted some of the various techniques used in cranial therapeutics and use manual muscle testing as a diagnostic aid. The evaluation and treatment techniques used in applied kinesiology are those which Goodheart²⁴ correlated

Introduction to Cranial Function

with muscle testing as the diagnostic tool. The motion of the skull accepted in applied kinesiology is generally that described by Sutherland.

The types of cranial therapeutics go under various names, such as osteopathy in the cranial field, craniopathy, cranial technique, etc. In the various approaches, there are both similarities and major differences in the movement concept and manipulative approach.

Various forms of cranial therapeutics use different systems of diagnosis to determine the need for treatment and, once administered, its effectiveness. Sutherland's osteopathic approach in the cranial field uses observation and very sensitive palpation to feel the motion of the primary respiratory mechanism. When dysfunction is located, mild pressure is applied to various locations on the skull. This approach may use motion inherent in the skull as a corrective mechanism, which acts through the dura to influence areas lacking motion. It may include exaggeration of the fault, direct corrective pressures, and separation of the sutures as some of the corrective procedures.

Some forms of cranial technique evaluate for symmetry of the skull, together with its general shape, and attempt to force the skull bones into an improved symmetry and more "normal" shape. This approach has created some iatrogenic problems. An attempt to make corrections when function is not

known can either create an involvement where none previously existed, or aggravate an existing problem.

Applied kinesiology's approach to cranial therapy depends on evaluation of the cranial mechanism by the use of manual muscle testing as introduced by Goodheart.²⁴ This approach of evaluating the primary respiratory mechanism with manual muscle testing indicates when abnormal function is present, how correction should be made, and, finally, whether the correction is successful in obtaining improved function. The system, when used by trained practitioners, does not appear to have iatrogenic effects and it produces excellent clinical results.

The efforts of the pioneers in cranial therapeutics provided invaluable information regarding skull movement and general knowledge of the primary respiratory mechanism. This background, combined with the applied kinesiology system of diagnosis, brings an easily learned and therapeutically effective tool to the physician. Without knowledge of the primary respiratory mechanism, its dysfunction and correction, there are many conditions that the physician cannot correct. In the absence of a therapeutically effective approach, the *symptoms* are usually managed by medication and other palliative approaches. If this approach is not effective, the condition will remain symptomatic and the true etiology will not be discovered.

Skull Motion

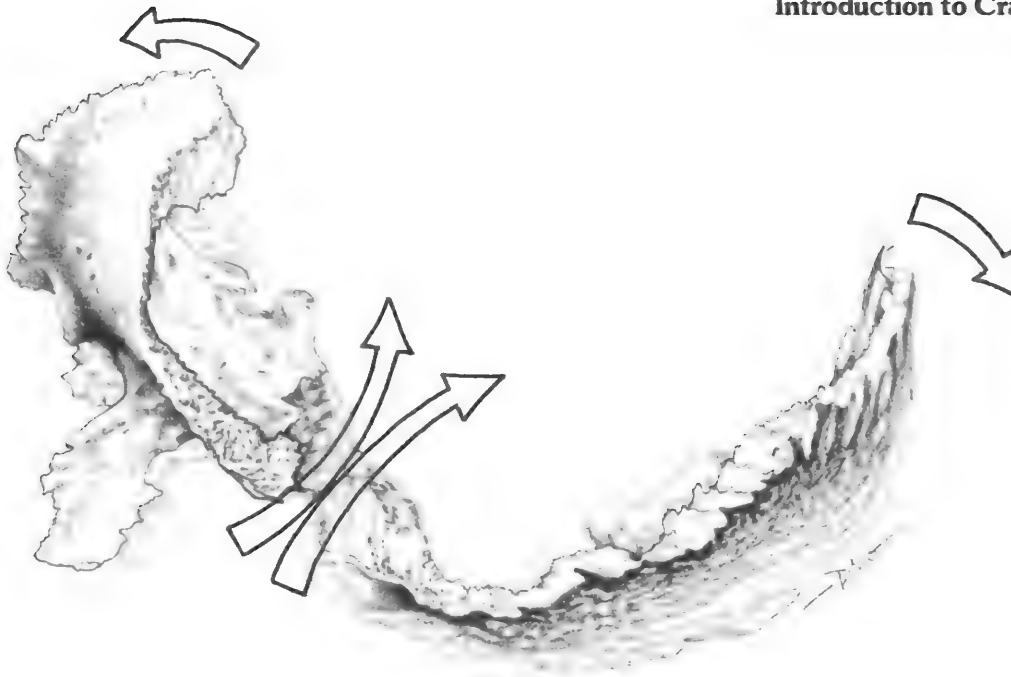
Skull motion, when normal, is specific and predictable. It was described by Sutherland in his text, *The Cranial Bowl*.⁶² General motion will be considered here and can be described by considering the sphenoid, occipital, and temporal bones.

The basilar portion of the occipital bone moves superiorly with the dorsum sellae of the sphenoid bone on inspiration. Inspiration can refer to either primary respiratory function or the respiration of breathing; this motion is called sphenobasilar flexion. On expiration, the sphenobasilar junction moves inferiorly.

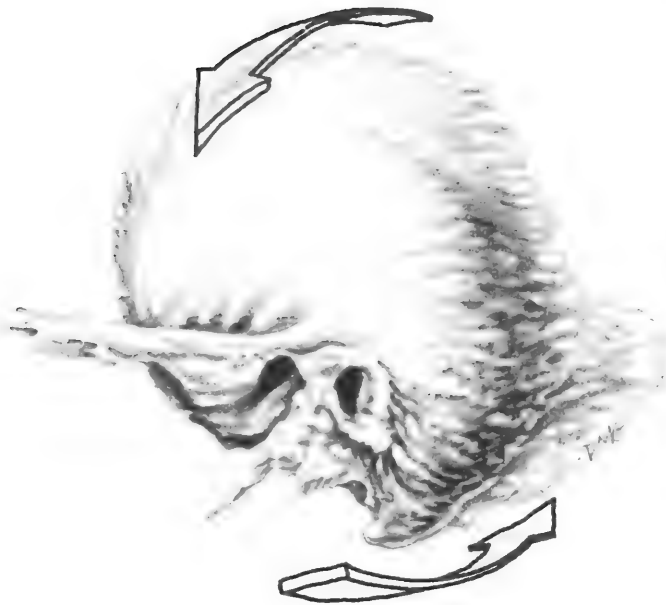
The temporal bone rotates in the sagittal and transverse planes. The general axis of temporal bone rotation is through the petrous portion. Because this rotational axis is approximately at a 60° angle with the squama, it causes a flaring of the temporal squama on inspiration. The superior portion of the squama moves anteriorly and laterally, while the tip of the mastoid process moves posteriorly and medially with inspiration. The opposite is true on expiration.

This general description of skull motion will be enlarged upon later in the anatomical consideration of the skull. The general motion described by Sutherland has been supported by findings in applied kinesiology examination and other research, which will be described.

To understand skull motion, it is first necessary to put into perspective what we are dealing with. Unfortunately, most anatomical studies of skull osteology are done on dry bone or cadavers. The role of the skull in most anatomy classes is perceived as protection of the brain and other nerve structures, and as something to "hang the face on." Some effort is usually made to discuss the entrance to the alimentary canal for assimilating and masticating food. Four of the five special senses are housed completely within the cranium, which adds to their function and protects them. What is not considered in most doctors' education is that the skull *in vivo* is a dynamic plastic structure which has additional organized activity. Studying the skull from a dried specimen or in a cadaver is similar to studying a

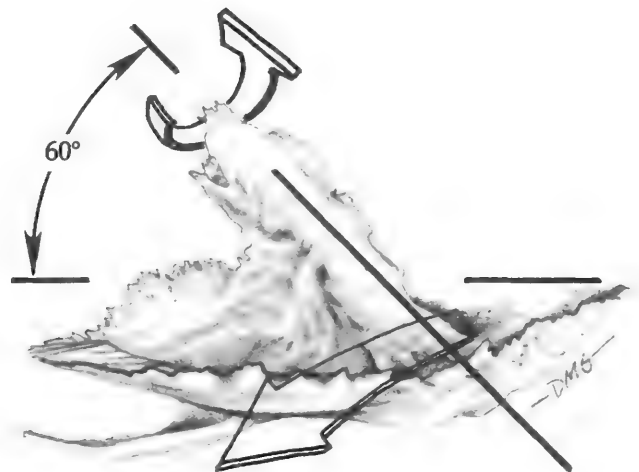


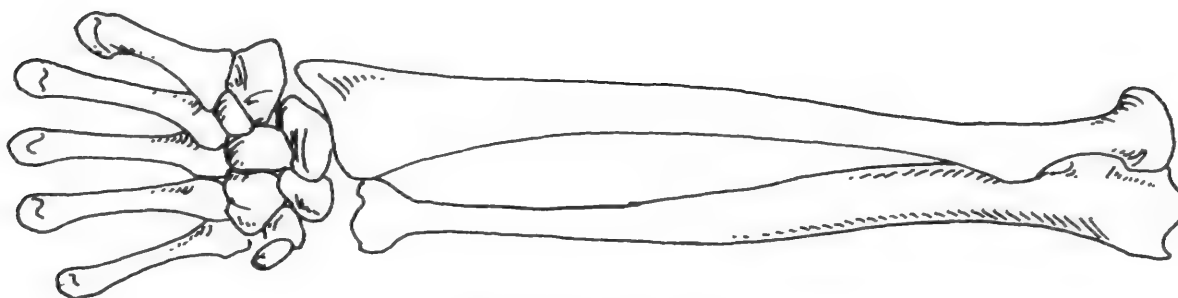
2—1. General motion of sphenoid and occiput on inspiration.



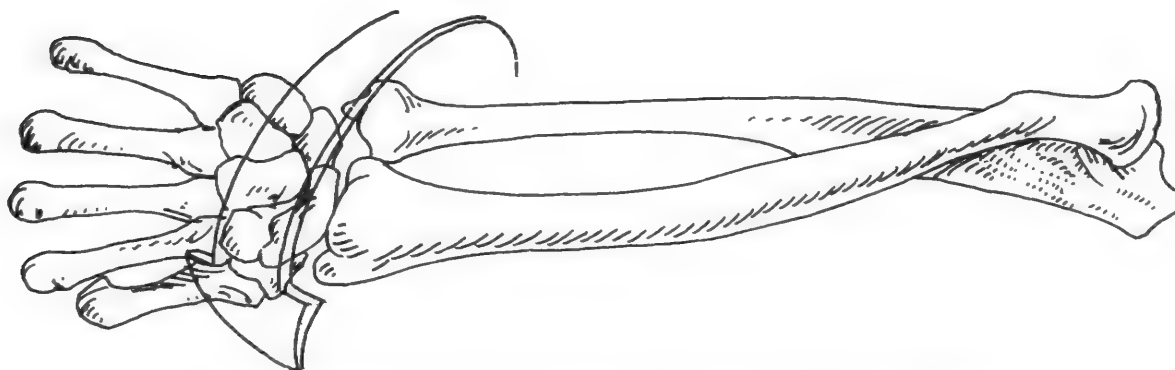
2—2. Lateral view of temporal bone indicating motion on inspiration.

2—3. Superior view of temporal bone showing general axis of rotation through petrous portion.

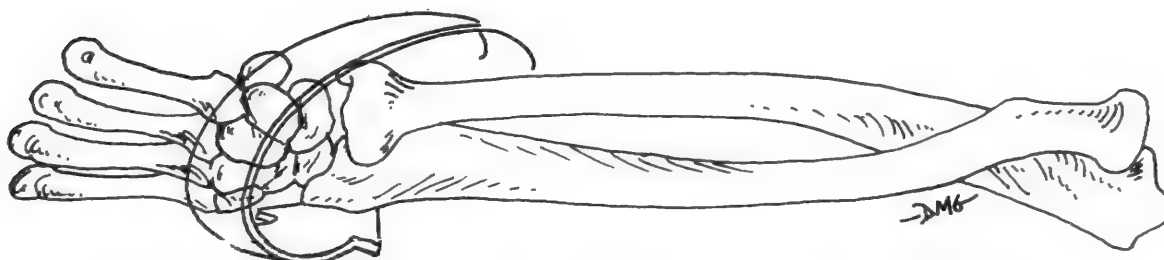




Right forearm in supination.



Pronation is limited in vitro because dry bone does not have flexibility.



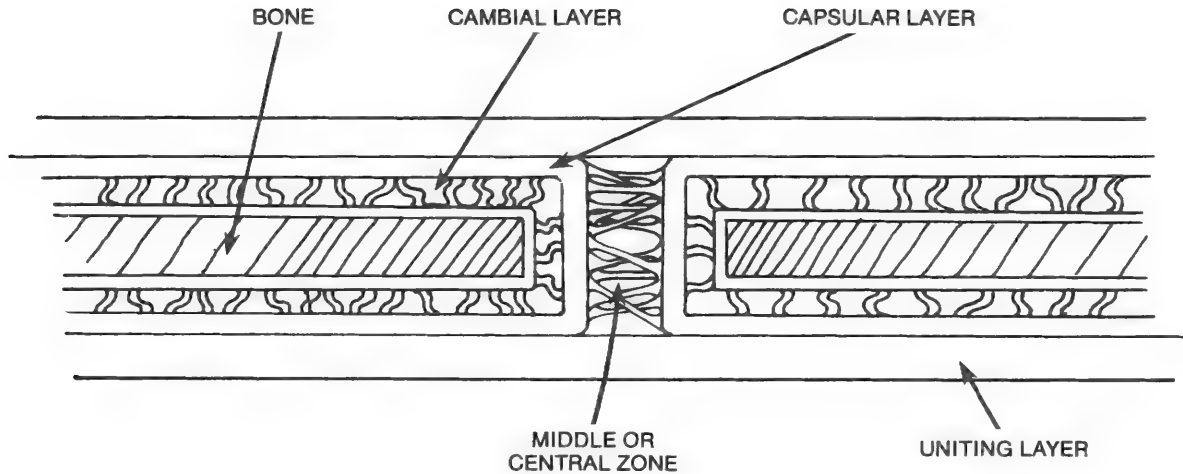
Pronation is greater in vivo (exaggerated in illustration). A torsion of 69° develops between the ends of the radius, and 34° between the ends of the ulna. After Stowe et al.⁵⁹

2—4.

telephone pole as if it were a living tree. The telephone pole is rigid, the sap has dried, there is no living flow, and an effort to bend it could cause it to break. A living tree, on the other hand, has water and nutrients in its “veins,” it bends and sways with the breeze, and it progresses through its life cycle from year to year.

The plastic nature of bone *in vivo* is illustrated in

the study by Stowe et al.⁵⁹ Orthogonal x-ray beams were used to measure the absolute and relative movements and consequent torsions of the adult human forearm *in vivo* during rotation of the forearm from its maximum voluntary supination to its maximum voluntary pronation. A torsion of 69° was observed between the ends of the radius; 34° was seen between the ends of the ulna.



2—5. Suture histology after Pritchard et al.⁵⁰

The consideration of bone flexibility is necessary when studying skull motion. As will be described later, there is motion at the sutures joining the bones of the skull, yet the motion of the total complex depends on the flexibility of the bone itself for normal function.

Keeping in mind the plastic nature of living bone, it becomes very obvious to the student that there is considerable flexibility in many areas of the skull. This is best observed on a disarticulated skull. The student can see that the bone is very thin in some areas, similar to parchment paper. Some of the structures making up the eye orbit and some of the areas of the sinuses have bone which is parchment-thin. Almost the entire ethmoid is this type of bony tissue. Some of the bone in the skull is highly cancellous, having a higher degree of flexibility than compact bone. The primary example of cancellous bone is the sphenobasilar junction, which is required to flex and extend to a small degree even after ossification is complete at about twenty-five years of age.

The nature of the sutures themselves indicates motion of the skull. Pritchard et al.⁵⁰ studied the histology of the sutures in human specimens and several types of animals. They found five distinct layers of cells and fibers between the edges of the adjoining bones. They concluded that "... the histology of the sutures suggests that it has two main functions, viz. that it is a site of active bone growth,

and that it is at the same time a firm bond of union between the neighboring bones, which nevertheless allows a little movement." The uniting layers are the strongest bond of union between the bones, and are evidently homologous with the fibrous capsules of diarthroses. There is a weak central zone of fiber bundles running in all directions, and sinusoidal blood vessels which could be regarded as analogous to a synovial joint cavity. They go on to state, "The great vascularity of the middle zone ... might simply be a means of filling the unwanted spaces between the fiber bundles without embarrassing movement; it might be a part of the emissary system of veins; on the other hand, it might serve as a hydrostatic cushion between the ends of the bone reinforcing the other protective measures against undue mechanical stresses."

Upledger et al.⁶⁷ studied specimens taken from living adult skulls at the time of neurosurgical craniotomy. Along with connective tissue, the sutures were shown to have the presence of viable myelinated and unmyelinated nerve fibers and nerve receptor endings.^{52, 53} These neurostructures can give sensory input to the nervous system, influencing the body in many and varied ways. The possible effect of impulses arising from the sutures and affecting the motor system of the body is an important consideration in the applied kinesiology approach to the cranial mechanism.

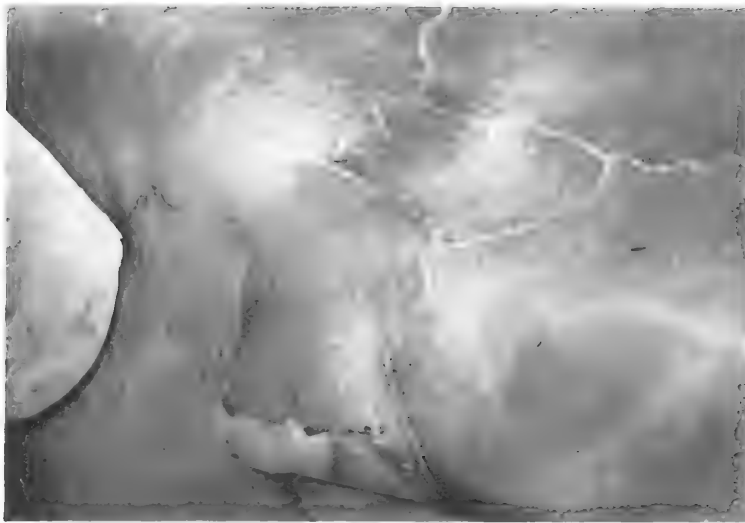


2—6.

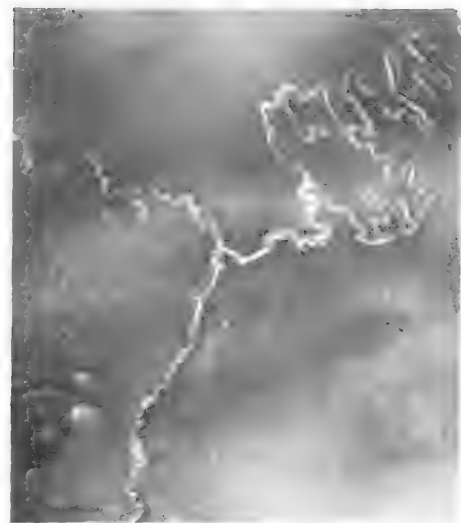
Transillumination reveals the thinness of the bones. Note the orbital area, greater wing of the sphenoid, and the squama of the temporal bone.



2—7.



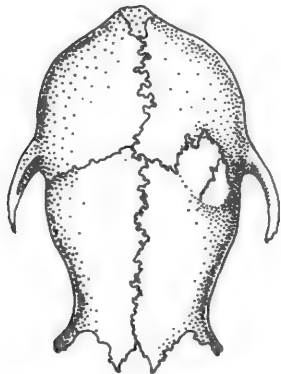
2—8. Transillumination through sutures of the pterion area. The thinness of the bony orbit is revealed at the left.



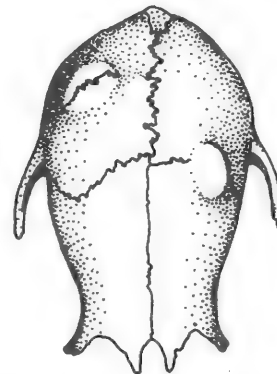
2—9. Light can be seen shining through the space between the sutures near the asterion.

An indication that there is a physiological need for the suture to be present for motion seems apparent from a study by Giblin and Alley.²² They attempted to determine what distortion of growth would take place as a result of surgically causing a suture to be fixed. The method was to cut a circular disc of bone from the coronal suture of three-week-old puppies. The circle was rotated *in situ* so the suture laid at an angle of about 90° to the rest of the suture. It was expected that bony union would take place at the cut edges around the entire circumference of the disc, causing fixation of the frontal and parietal bones, and that there would be continued growth at the suture resulting in a buckling and an elevation of the two halves of the disc. What actually happened was the development of an artificial suture along the cut edges to maintain continuity of the coronal suture.

In another study done by the same team, one disc was cut from the coronal suture and one from the parietal bone. The parietal disc was placed into the coronal suture area, and the coronal suture disc into the parietal bone. The coronal suture placed into the parietal bone remained open on the outer surface only, while the solid disc from the parietal in the coronal suture caused a fusion of the frontal and parietal bones. Here it is interesting that the coronal suture in the circular bone placed into the parietal bone began to fuse, whereas it did not when it was simply rotated *in situ*. They stated, "Sutures fuse by complete ossification of the membranous tissue. A primary condition for this to occur (given normal hormonal and nutritional factors) appears to be absence of movement: compare the union of fractures, and the synostosis of immobilized joints. In the case of most of the cranial sutures there must be



2—10. Development of new suture at cut edge. After Giblin and Alley.²²



2—11. Exchange of bone discs between coronal suture and parietal bone. After Giblin and Alley.²²

Introduction to Cranial Function

cessation of growth in all parts of the skull before they can fuse. Conversely, if premature fusion occurs at any point, it will tend to spread progressively due to the rigidity produced.” Another finding in their study was that the surgical fixation of the coronal suture caused rapid fusion of other parts of the suture.

Studying the sutures in aged monkeys, Retzlaff et al.⁵² found that sutures prevailed even in the oldest monkeys in the study. The ages of the monkeys ranged up to twenty years. This disagrees with previous comments⁴ which indicated that the sutures disappear shortly after the brain is fully grown, which occurs in young animals. The study of Retzlaff et al. indicated that if there is premature ossification of sutures, it occurs as a result of pathological change rather than aging.

Bolk⁴ studied the sutures of 1,820 non-adult skulls. He found the most common suture to close prematurely to be the occipitomastoid. This is an area of very important motion in the cranial primary respiratory mechanism. The frequency of wormian bones in this area seems to indicate an effort of the body to maintain motion when it is restricted. Since his group of skulls ranged in age from three to twenty years, he was able to determine that frequency of closure of the occipitomastoid suture does not increase after six or seven years of age; the premature obliteration of this suture is limited to infancy, beginning as a rule before the end of the sixth year. An interesting aspect of Bolk's study was that of the total group, there were 343 skulls (19%) which showed either one or more premature suture closures. This high percentage is largely due to the frequent premature closure of the occipitomastoid suture, which numbered 272 cases of the 343 with early closure. The question must arise as to the circumstances whereby this large collection of young skulls was obtained. Obviously, death was not due to old age. Were the individuals in poor health which caused death? If so, according to cranial primary respiratory principles, it would be expected that the skull would not have normal motion, and there would be possible early closure of the sutures.

A very interesting additional aspect of Bolk's study is the correlation of early suture closing and the persistency of the metopic suture. In skulls of Dutch origin, where the collection came from, the persistence of the metopic suture is found in just under 9%. Interestingly, of the 272 skulls with premature closure of the occipitomastoid suture, there were only two cases which had a persisting metopic suture. Relating the usual 9%, there should have been 24 with a persisting metopic suture; there were only .7%. Again, in the philosophy of the cranial

primary respiratory mechanism, a very mobile skull would tend to have a persistent metopic suture. A skull with limited motion would have a tendency for closure of the sutures, including the metopic suture as well as the occipitomastoid.

In applied kinesiology, one of the primary principles of the cranial primary respiratory mechanism is that balanced muscle action is necessary for normal cranial function. This is particularly important in the muscles of mastication. Washburn⁶⁹ unilaterally removed the temporalis muscle from rats to observe the changes that would take place in the mandible and cranium. He noted a change in sutural growth on the disturbed side. In some areas, the suture failed to develop the normal complexities, while in other areas it became very complex. This indicates that where the loss of muscle caused decreased growth, sutures were simpler than normal; where growth was prolonged, sutures became more complex.

The architecture of the sutures is consistent with skull motion. Close study of a disarticulated skull reveals squamous-type sutures where sliding action is necessary, serrated sutures for expansion and contraction or for a gear-type motion, and occasionally solid bearing-type articulations. The most interesting observation is the change of sutural shape where there is a general axis of rotation of skull function. It seems illogical that anyone could make a detailed study of the sutures of a disarticulated skull and not conclude that the skull is designed for motion, minute though it may be.

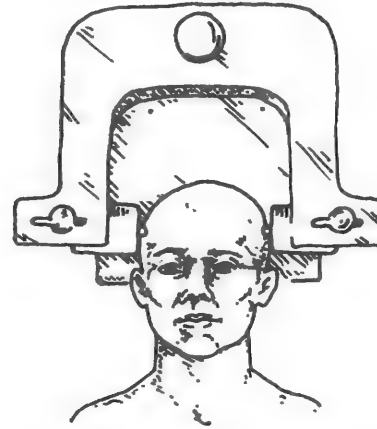
Two simple demonstrations described by Magoun⁴⁵ can be done on one's own skull. These demonstrations not only reveal that motion is present; they provide an opportunity for the initiate in cranial function to feel the subtle motion present in the skull. Place the thumb and forefinger, one on each frontal process of the maxillary bone. Press with the tongue against the palate next to the dental arch on one side and then on the other. Discretely feel for motion of the maxillary at the frontal process. The second procedure is more difficult to do correctly. Press bilaterally in a medial direction on the zygomatic bones. After a few seconds of pressure, take a breath through the nose and feel the restriction of air flow. Care must be taken that the soft tissue isn't being pushed medially, but rather just the bones. If the nasal passages were clear in the first place, this compression of the structures will reduce breathing capability. Of course, it is important that the cranium be functioning in basically a normal manner or these demonstrations may not reveal the motion present in the skull.

Objective Measurement of Motion

An important study of cranial motion was done by Frymann.²⁰ She used mechanical transducers designed to measure small amounts of motion and recorded the motion of the skull on a strip recorder. Her goal was to determine if skull motion was actually present and, if so, with what the motion correlated. The transducers were mounted on either side of a yoke, fitting around the subject's head in such a manner that each transducer contacted a side of the head. The yoke was firmly attached to a table, and the electrical circuit of the transducers was such that if the subject did move, one transducer would give a positive signal and the other a negative one, thus cancelling each other. If there was expansile movement of the skull, there would be a positive signal from both transducers recorded on one channel of the strip recorder. The transducers were adjustable to the skull to contact at various locations in different testing procedures. In these procedures, contact was made on the dermis of the tissue overlying the cranial bone being measured. Pressure of the contacts into the tissue caused an expression of the fluids from the interstitial spaces, after which a re-tightening of the probes was done. The other available channel recorded either phethysmography or pneumography.

The first successful recording of skull motion was done May 30, 1963; it seemed to correlate with Sutherland's contention of cranial primary respiratory cycling. This cycling of apparent cranial motion continued when the breathing respiration was held in the inhalation phase, and was slower than the breathing respiratory rhythm preceding and following.

Further studies revealed a correlation to other cycling factors of the body. By using a two-channel strip recorder, the breathing and pulse wave of the subject were compared with the motion of the skull. During pulse and cranial recording, one channel was intended to record cranial motion and the other the pulse. Because the cranial transducer had the soft tissue of the skull interposed between the probe and the bone, there was a small pulse recording picked up on that channel as well as on the channel intended for plethysmography alone. The pulse wave recorded from the cranial force transducer decreased after the initial adaptation of the tissues to the pressure of the probe, and after the probe was tightened. Standard plethysmography of an extremity was fed to the other channel of the recorder. The pulse wave observed through the force transducer on the skull paralleled that of the plethysmograph, and in addition the base line of the plethysmograph varied with apparent cranial motion. This seems to show that a wave correlating with cranial motion is also present



2—12. Study of motion with fixed transducers. After Frymann.²⁰

in distal body function, or, in other words, there is a general pulsation throughout the body that is of greater intensity in the skull.

The primary respiratory motion often continued throughout breathholding, but its characteristic sometimes changed. In some instances, deep inspiration significantly changed the characteristic of the skull motion. On some recordings, comparing the wave from the skull activity with a pneumogram showed paralleling of the two activities, followed by a change to independent cycling.

One of the most interesting aspects of this study is the small amount of motion measured, from 0.0005 to 0.001 inch. The exact amount of motion must be questioned because of the inability to exactly quantitate the measurement due to the compressible soft tissue between the transducer probe and the skull bones.

Although Frymann made an effort to study and report on normal skulls as observed by osteopathic cranial palpation, it should be emphasized that there was a variable in the measured response of skull function. In some instances, the cycling of the skull would vary with the subject's breathing respiration, while in others it was parallel. It seems reasonable to question whether there were varying forms of normality of the skulls of the subjects evaluated because of the study's variable results. Frymann's efforts have contributed to the understanding of the cranial primary respiratory mechanism, but additional studies are needed to further evaluate normal skull motion and the various forms of examination systems and therapeutics.

Frymann's study was the first to provide objective information regarding skull motion which did not require an examiner's interpretation of his palpatory

Introduction to Cranial Function

art. From this study it appears that the cranial primary respiratory mechanism is an autonomous cycling which is influenced by the subject's breathing respiration.

In an effort to eliminate the possibility of artifacts from the intervening tissue between the transducer probe and the skull, Michael and Retzlaff⁴⁶ attached force transducers to a screw eye placed in the parietal bone of anesthetized monkeys. Each animal's head was secured in a stereotaxic frame. Blood pressure was monitored at the femoral artery or by a cannula advanced to the heart. Respiration was monitored by either tracheal pressure change or thoracic displacement.

A cyclic cranial bone displacement of 5 to 7 cycles per minute was observed, which could not be attributed to either respiration or heart rate. There was another rhythm present which corresponded to respiration. Movement of the parietal bone was also recorded upon spinal flexion and extension and from direct application of force to the cranium. The movement of the parietal bone upon spinal movement supports Sutherland's contention of sacral primary respiratory movement connected with the cranium by the dura. The movement of the parietal bone by external force seems to indicate that trauma or therapeutic pressures can change the cranial bone relation.

In an accompanying paper, Retzlaff et al.⁵¹ hypothesized that the movement of the monkey's body changed the cerebrospinal fluid pressure, thus influencing the motion of the skull bones. The maneuvers of the monkey's vertebral column caused both the slow and the fast oscillatory waves to increase in amplitude. Sutherland's theory of cranial bone movement was that the cerebrospinal fluid pressure changes caused by brain movement are responsible for the spontaneous bone movement.

Recordings of skull motion were obtained by surgical sutural material taped to the frontal and mastoid regions, and then tied to force transducers. This study by Tettambel et al.⁶³ demonstrated skull movement independent from simultaneous pulse and respiration recordings, revealing the cranial rhythmic impulse as a distinctive wave and not a component of pulse or respiration. The study included thirty subjects ranging in age from sixteen to seventy-one. The subjects were evaluated with osteopathic palpation; a correlation was observed between the side of body trauma, palpation, and recorded cranial activity.

X-ray has not been routinely used as a diagnostic method in evaluating for cranial dysfunction. One such study, done by Greenman,³⁰ was correlated with the clinical findings of an osteopath trained in the cranial concept. It appeared from this study that

it is possible to demonstrate side-bending, torsion, and flexion and extension patterns of the skull on x-rays. There was also a correlation of low sacrum and low occiput on the same side.

Evaluation of skull change following osteopathic cranial manipulation was done with dental procedures by Baker.² Serial measurements were made on models of the maxillary teeth, and treatment consisted of occlusal equilibration by the dentist and osteopathic adjustment for six months. This revealed a change of 0.0276 inch increase in the distance between the second molars, which is nine times the possible error in his measurement technique. It was concluded that the patient's skull bones moved along their sutures. This rather gross change has significant bearing on the changes of occlusion following trauma or cranial therapeutics. It is often necessary to obtain dental consultation and equilibration for permanent correction of skull dysfunction. There are many other ramifications of skull motion correlating with dental procedures which must be considered by both the cranial therapist and the dentist. These will be discussed more thoroughly in the section of the book dealing with the temporomandibular joint and dental procedures.

A method of evaluating dental occlusion is to record the sound as the teeth meet in intercuspation.⁵⁷ This recording is called an occlusogram, and it records alpha and beta components. An ideal occlusion consists entirely of the beta component. The alpha component is a relatively high frequency signal caused by the teeth slipping and sliding together as a result of prematurity. The quality of the occlusion can be observed by a trained individual listening with a stethoscope to the sound of the teeth meeting; an individual knowledgeable about occlusion can evaluate the sound on himself. Silverman⁵⁵ states the audible, sharp, staccato-like sounds of his own normal occlusion change under different circumstances. After sleeping in a specific position which placed imbalanced pressures on his skull, he would awaken with a defective occlusal contact producing both the alpha and beta sounds he personally observed by clicking his teeth together. The deviation from normal occlusion would last from several minutes to over one hour. This experience prompted him to make a continual evaluation of his occlusion and the factors which would change it. He found that pressing on various areas of the skull would temporarily decrease the ideal occlusion into one of prematurities. While the imbalanced occlusion would last for varying lengths of time, he observed that swallowing would usually immediately return the occlusion to normal. He later found that deep inspiration was even more effective in returning the

occlusion to normal. Silverman further found that patients sitting in a dental chair with the head resting in a V headrest experienced a change in occlusion, and under these circumstances he was equilibrating to an improper balance of body activity. As a result, he developed a procedure whereby the patient would take three deep breaths with his head unsupported prior to evaluation of occlusion. Not only does this give evidence of cranial bone motion; it also shows the importance of considering the cranial primary respiratory mechanism in the fine art of equilibration of the teeth. Henningsen^{31, 32} points out how the V-shaped headrests of some dental chairs make contact with the mastoid portion of the temporal bone, influencing cranial position. This will be discussed further in the section of this text dealing with occlusion.

Skull flexibility and its influence on occlusion are regularly observed on a clinical basis. It is seen dramatically on individuals with a maxillary dental splint or upper dentures. As cranial corrections are obtained, the splint which originally fit closely over the teeth of the maxillary arch will no longer seat correctly. Under these conditions, if the splint is forced over the teeth and the patient bites down, the cranial faults will probably return. The same is applicable to the upper dental arch when the patient wears an upper denture, although it is not as severe because of the gingiva's compressibility. It will often be seen on an upper denture on which the palatal area is asymmetrical. Here the change in position of the palatine and maxillary bones as a result of cranial corrections may cause the denture to no longer fit. (Appliances and dentures are discussed more thoroughly in the second section of this text.)

There are physical factors which occur in the body with cranial primary respiratory motion for which the data base is not adequate to explain. Upledger and Karni^{65, 66} monitored subjects for electrical and physical changes of the body during osteopathic cranial diagnosis and treatment. They used mechanical transducers to record body motion and recorded electrical activity by electrocardiograph and electromyograph electrodes applied to the body. These factors were compared with what is termed a "release" of the skull as observed by an osteopath trained in the cranial concept. The release is described as physician-perceived sensations that range from a smooth, rhythmic motion which ebbs and flows and occasionally ceases entirely, to a quick, jerky or vibratory motion which may be quite irregular. The diagnosing physician reported blind his sensations of the skull to the observer of the physiologic parameters. Although each individual evaluated had unique personalized patterns, there

were repetitive features in all the tracings which allowed the consideration of four different patterns: (1) rapid oscillations, (2) transient wave forms, (3) rapid wave forms or spikes, and (4) base line changes. The movements of the skull as perceived by the examining physician were classified as (1) normal rhythm, (2) stillpoint, (3) end of stillpoint, (4) release, (5) shifting, which is the impression of a tidal fluid motion and changes of direction of flow as perceived by the examining physician, (6) pulsating, a rapid oscillatory motion of low amplitude and high frequency (50-80 cpm), (7) wobbling, a lower frequency (20-40 cpm) than those of pulsating, and (8) torsion, a sensation felt by the examiner of a rotational periodic motion about a longitudinal axis through the patient's body where all sections of the body are not moving synchronously.

As the examining physician reported the skull's activity as perceived by palpation, the observer noted that specific electrical and mechanical patterns correlated directly with the subjective impressions of the specific changes perceived by the physician.

Electrical phenomena about the scalp were observed by Girton et al.²³ The very slow wave form was observed during the development of improved scalp electrodes for use with direct, coupled amplifiers. This slow wave is filtered out of conventional electroencephalograms due to the short time constant of the resistance-capacitance coupling used in the amplifier system. The waves were generally 5 to 8 cycles per minute, and were not always present. In some instances, an individual would show no slow waves during one recording session, but exhibit them at another time. In other instances, the wave would start or stop during the recording session. In some instances, the wave would be simultaneous with respiration but continue when respiration was held. The presence or absence of the wave could not be predicted, nor could it be started or stopped at will by various methods attempted. No attempt at cranial bone evaluation or manipulation was done. It appears that this may be a fruitful area for further study in reference to possible correlation with cranial primary respiratory function.

Rhythmic motion of the brain, skull blood flow, available oxygen, electrical phenomena, etc., have been observed in research which was not designed to study the cranial primary respiratory system.^{1, 7, 23} It is possible there is a relation of these various types of activity with the cranial primary respiratory system. Further research is needed to study these questions.

Most of the research on the cranial primary respiratory system has been accomplished by the osteopathic profession. The supportive evidence of this motion, as described by Sutherland, is quite

Introduction to Cranial Function

convincing. It now becomes necessary for the various professions working with the cranial concept to research their own approaches to this system. As the data base grows, there will be a clarification as to the etiology of many health problems which are

currently considered to just develop in susceptible individuals. When the basic underlying causes of health problems are understood, they are correctable conditions rather than conditions which are symptomatically controlled.

Role of the Cranium in Health and Dysfunction

The evidence of skull motion correlating with the primary respiratory mechanism described by Sutherland⁶² is indeed strong. The importance of this activity is negligible if disturbance in the normal pattern does not create health problems. Here we will briefly introduce the various ways that cranial dysfunction is believed to cause disturbance in body function. These will be discussed in more detail as the anatomy and the physiology of the stomatognathic system are presented.

Cranial Nerves

Entrapment neuropathy is considered to be a localized injury and inflammation in a peripheral nerve which is caused by a mechanical irritation from some impinging anatomical neighbor.³⁶ Within the cranium and at the foraminal openings there are innumerable areas where the nerve can be impinged from tortuously traversing bony ridges, by bony foramina, and by soft tissues. Dysfunction as a result of peripheral nerve entrapment is well documented in the literature.^{36, 48} It has been shown that the dorsal spinal roots are more susceptible to compression block than peripheral nerves. For example, the dorsal root requires only 20 mm Hg of pressure to produce the same conduction block which is present by 100 mm Hg to the sciatic nerve when pressure is applied for three minutes, followed by three-minute recovery periods.⁵⁴ It is not known whether the cranial nerves possess decreased resistance to compressive forces as they traverse areas of possible compression, as does the spinal nerve within the intervertebral foramen.

The mechanism interfering with normal nerve transmission may not be a block or hyperirritability. In a classic experiment, Granit et al.²⁹ demonstrated the creation of fiber interaction within a nerve from compression. This artificial synapse was produced by a pressure so gentle that it did not impair conduction of the original impulse. Another important factor of this investigation was that the nerve returned to normal after being decompressed and irrigated with a saline solution.

Nerve Receptors Within Sutures

Headaches and facial pain⁴³ may result from

jamming of the sutures, which causes an increased stimulation to the nerve endings within the sutures as described by Retzlaff et al.^{52, 53} Abnormal stimulation of these nerve endings also appears to cause interaction in the neuronal pools, influencing many aspects of the nervous system. This is observed with applied kinesiology evaluation by challenging the skull and evaluating muscle strength change by manual muscle testing. When the skull is not functioning normally, the nerve receptors in the sutures appear to be abnormally stimulated by the challenge, thus influencing the muscle test. Retzlaff et al. recommend diagnosing a temporal headache by either separating or jamming the suture with digital pressure to either relieve or increase head pain.⁵³ The challenge mechanism of applied kinesiology is similar to this, but it evaluates influence throughout the body rather than just the localized pain.

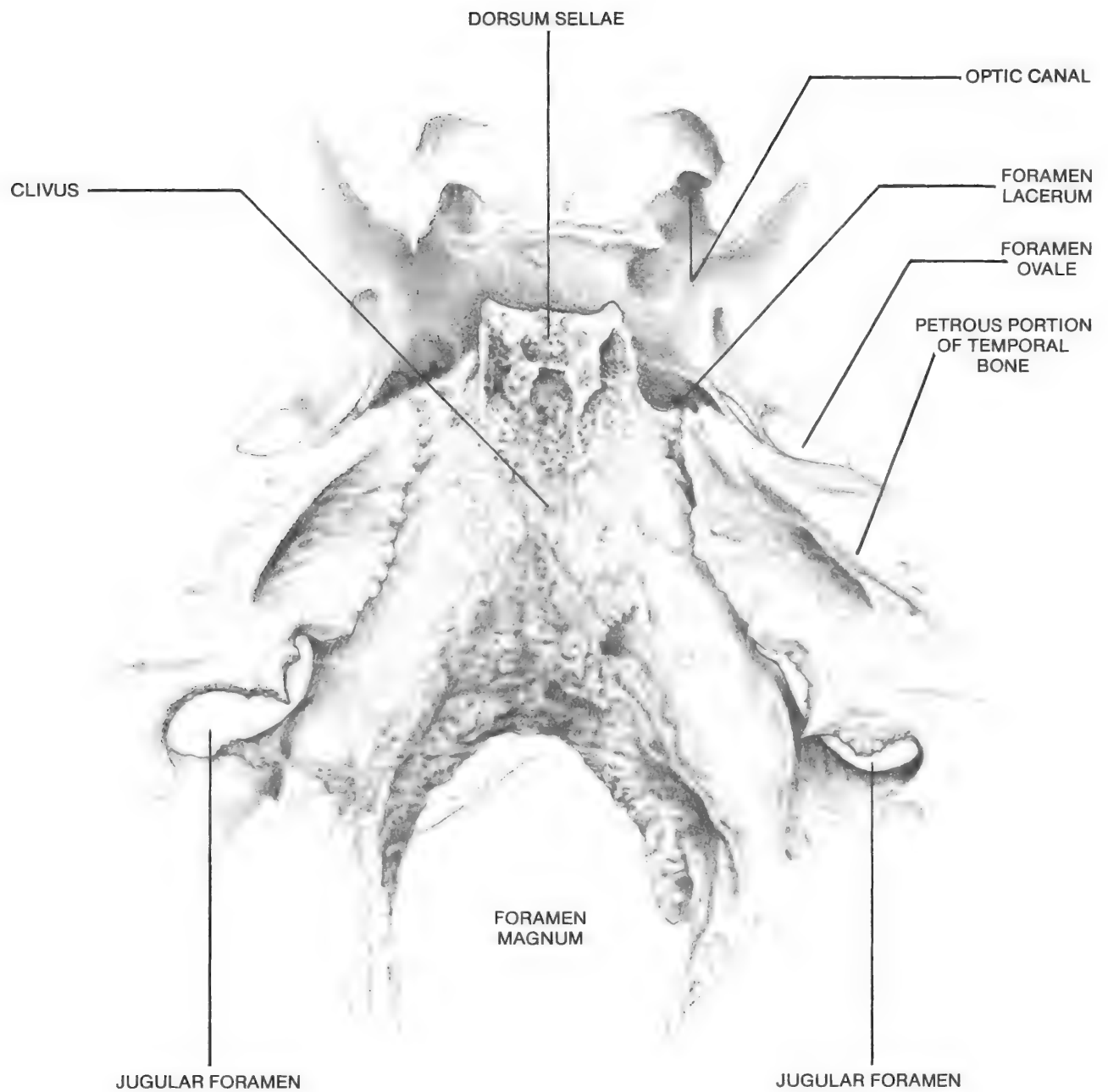
Cerebrospinal Fluid

It is believed that the cerebrospinal fluid has a rhythmic fluctuation with that of the skull. This fluctuation is the movement of the cerebrospinal fluid rather than actual circulation within the system.⁴⁵ It is also hypothesized that cranial motion has a direct relation to the pressure maintained in the closed cerebrospinal fluid system. It is important to both the mechanical support and physiology of the central nervous system, and is thought to carry secretions of the pituitary gland.⁴⁰

It is possible that the cerebrospinal fluid has a much greater function than that which is commonly cited. Erlingheuser¹⁸ proposes a theory about the circulation of the cerebrospinal fluid through hollow collagen fibers of the connective tissue system. He considers this a second circulatory system within the human body.

Steer and Horney⁵⁸ demonstrated cerebrospinal fluid flow from the subarachnoid space along the spinal nerves. The flow was interrupted by ligature of the nerve. Somberg⁵⁶ found that under low pressure cerebrospinal fluid passed only to the spinal ganglion.

These factors which are a matter of controversy indicate there may be many significant ramifications of the cranial primary respiratory mechanism which



2—13. Illustration of aged edentulous skull. In the cranial primary respiratory concept, there is major motion of the petrous portion. If disturbed, it may cause peripheral nerve entrapment at the jugular foramen or foramen lacerum.

Introduction to Cranial Function

are as yet undiscovered. Clinical results from correcting cranial faults indicate that much is unknown regarding the physiology of this mechanism.

Blood Pressure

Idiopathic hypertension frequently appears to be caused by disturbance in the cranial primary respiratory mechanism. The hypothesis for the cause of the elevated blood pressure that responds to cranial correction is that there is a reduction of cerebrospinal fluid pressure because of the cranial faults. Through baroreception the body recognizes a need for increased cerebrospinal fluid pressure and elevates the blood pressure, increasing the pressure in the venous plexuses and sinuses of the cranium, thus causing a secondary increase of cerebrospinal fluid pressure. The brain and spinal cord are within a closed compartment. The total fluid content of the three types — arterial, venous, and cerebrospinal — must be constant; otherwise pressure changes will result. When the arterial blood pressure rises, there is no change because the venous sinuses and plexuses, having a low pressure, take up the extra pressure. On the other hand, when the venous pressure increases, there is an immediate rise in cerebrospinal fluid pressure because the arteries, having a higher pressure, are not compressible to take up the extra pressure from the venous pressure.³ Correction of the cranial fault often causes an immediate lowering of the blood pressure. Of course, this is treating the basic underlying cause of the condition rather than the effects. An analogy can be drawn in the presence of arteriosclerosis or atherosclerosis. The body will automatically raise the blood pressure to obtain better circulation. Lowering the blood pressure by chemical means is not treating the primary cause. Treating the arterio- or atherosclerosis is the approach to the primary cause.

Circulation to the Brain

Normal function of the brain requires excellent circulation. The brain's oxygen requirement is indicated by the fact that the oxygen level is lower in the venous blood from the brain than from any other organ except the heart. The body uses approximately 250 ml of oxygen per minute when at rest, of which the brain uses 57 ml per minute; the heart only uses 22 ml per minute. The brain, comprising only 2% of the entire body weight, uses 20% of the total oxygen uptake. The brain even uses the same amount of oxygen during sleep.³³

Circulation to the brain is primarily determined by two opposing sets of forces: (1) the level of the blood pressure, and (2) cerebral vascular resistance. Arterial blood pressure can be within wide ranges

and have normal circulation to the brain. Factors influencing resistance are intracranial pressure, blood viscosity, and vascular diameter.³

As will be seen later, many of the blood vessels within the cranial cavity are in a position to be encroached upon by sharp bony ridges, edges of soft tissue, or tortuous pathways. It seems reasonable that an increase of cerebrospinal fluid pressure from cranial dysfunction could possibly cause vascular resistance.

It has been stated that the restriction of venous drainage from the skull influences cranial circulation.³⁹ Before this can be accepted, direct studies of brain circulation in relation to cranial disturbance must be done. Moyer et al.⁴⁷ demonstrated that there was no effect on cerebral circulation when the jugular vein was compressed by a tourniquet placed around the neck to reduce blood flow from the cranium. The restriction was increased to the point that the jugular (bulb) pressure was raised to 124-300 mm of water. They proposed that the brain's circulation would not be affected until the pressure of 450 mm of water is reached, which is the amount the cerebrospinal fluid must reach before cerebral blood flow is depressed.³⁵

Cerebral vascular resistance is most influenced by the diameter of the cerebral blood vessels.³ It seems probable that mechanical entrapment or encroachment from cranial dysfunction can influence circulation.

The exact mechanism of vasomotor regulation in the brain is not known. It is significantly influenced by concentrations of carbon dioxide.³

The importance of normal circulation to the brain is emphasized by that organ's dependence on oxygen from the blood it receives. The brain uses 20% of the oxygen consumed by the body; however, it receives only 15% of the total cardiac output.³³ If there is restriction of the cranial circulation, the limited oxygen availability may well cause functional disturbances manifested in many areas of the body that go unrecognized as to the basic underlying cause, which may be disturbance in the cranial primary respiratory system.

Endocrine Disturbances

The endocrine system may be disturbed in many ways by dysfunction of the cranial primary respiratory mechanism. Magoun^{40, 45} cites Sutherland as indicating that "... the inactivity of the pituitary body in the sella turcica through mechanical membranous articular restriction is the primary cause of pituitary secretory disturbances." This is clinically supported by many investigators.^{5, 28, 60} Goodheart has also described clinical evidence indicating direct influence of cranial dysfunction on the pineal gland.^{26, 27}

The endocrine system may be indirectly influenced by cranial nerve entrapment as a result of cranial primary respiratory dysfunction. Inasmuch as the endocrine system is an integrated system, one gland operating improperly may upset the entire system on a functional basis. Many who specialize in the endocrine system do not recognize the subtleties of functional endocrine disturbance where frank pathology is not present. Although these disturbances are not disease processes, they are directly responsible for many of the conditions people experience that are diagnosed as psychosomatic. A thorough discussion of the applied kinesiology approach to the endocrine system is in Volume V of this series.

Visual Disturbances

There appear to be two ways that visual disturbances result from cranial faults: (1) the change in shape of the bony orbit creating pressure on the eyeball, and (2) disturbance to the nerves controlling the extrinsic muscles of the eye. There may also be disturbance from entrapment of the optic nerve.

The bony orbit is made up of seven bones — the frontal, zygomatic, sphenoid, ethmoid, lacrimal, palatine, and maxillary. The orbit's shape can be altered by change of bone position from sutural movement and/or from the flexibility of the living bone. The medial wall of the orbit especially is exceptionally thin-boned, much like parchment paper. A change in the shape of the bony orbit can alter the shape of the eyeball. This change can disturb the distance from the lens to the retina, altering visual acuity. Often, correcting cranial faults will immediately improve visual acuity.

The nerves to the extrinsic muscles of the eye can be disturbed by cranial faults which cause muscular weakness.²¹ The weakness of these inaccessible muscles is very similar to that observed in postural muscles throughout the body by applied kinesiology evaluation. Special testing procedures are used to evaluate the balanced function of these muscles. Generally the condition is functional, causing the eyes not to function optimally but there is no diplopia present. In applied kinesiology, this has been termed "ocular lock" (see Volume I). In most cases, failure of the eyes to function together is not observed by simply looking at an individual, unless the examiner is well-trained.

Auditory

Disturbance in hearing in the form of loss or tinnitus can develop as a result of cranial faults. There is also a correlation with the vestibular portion of cranial nerve VIII, which interferes with equilibrium

and posture and will be briefly discussed next.

Neurologic Disorganization

Relating to the cranial mechanism are many proprioceptors which must integrate for the balance and equilibrium mechanism of the body. The visual righting reflexes, labyrinthine reflexes, and neck righting reflexes must all integrate and are very dependent upon normal cranial function. In addition, the sternocleidomastoid and upper trapezius receive dual nerve supply from the spinal accessory (cranial XI) and spinal nerves. If there is inappropriate correlation between these different nerve supplies, confusion which manifests itself as neurologic disorganization (switching) is observed through applied kinesiology evaluation. Cranial dysfunction is suggested as an etiology for idiopathic adolescent scoliosis by Magoun.⁴¹ The condition has been correlated to neurologic disorganization by Goodheart.²⁵ Close observation will reveal that nearly all cases of this problematic condition will have both neurologic disorganization and cranial dysfunction, the two factors probably contributing to each other. (See Volume IV for complete discussion.)

Temporomandibular Joint Dysfunction

Much has been written about temporomandibular joint dysfunction in recent years. It relates to occlusion of the teeth and muscular balance. The cranial mechanism is also involved in an interdependent function. Mandibular function and occlusion must be normal or adverse influence on the cranial primary respiratory mechanism will result. On the other hand, cranial faults can disturb the occlusion and mandibular function. The ramifications of abnormal temporomandibular joint function are extremely wide and are discussed in the second and third sections of this text.

Holographic Concept

Current knowledge of the way the brain controls body activity is very limited. This limited knowledge is also true of the cranial primary respiratory system. Although there is definite evidence that the skull has physiologic motion which is necessary for normal body function, its relationship with the nervous system and other controlling patterns of the body is mostly hypothetical. It is obvious that as knowledge develops in general neurophysiology, neuropsychology, and the newer field of psychobiology, concepts and understanding of this subject will change drastically.

It is important to understand that most of the current functional concepts regarding the brain and cranium are hypotheses, although some have devel-

Introduction to Cranial Function

oped to the level of theories. As these hypotheses are tested, they will need to be changed and remodeled to fit current knowledge.

Karl Pribram of Stanford University has developed an analogy that the brain stores information in a manner similar to a hologram. A holographic image is recorded by a single source of coherent light from a laser which is split into primary and secondary reference beams by a mirror. In a holographic recording the image is three-dimensional and all parts of the image are recorded on the entire plate. Unlike a standard photograph, when a hologram is cut in half there are two complete images, just smaller in size and with less detail. When a standard photographic image is cut in half, part of the image is on one piece and the remainder is on the other. The entire image can be seen only by reuniting the two halves. Recording information in the brain as a hologram is compatible with Pribram's findings that portions of the cerebral cortex can be removed from monkeys and, no matter which area is removed, the monkey can still perform a learned task. Memory is only destroyed when too much of the brain is removed. Pribram found that neither muscles nor movements are

represented in the cortex, but rather actions.

Applying the current concepts of brain function, Goodheart has proposed a hypothesis that the cranium acts as a parabolic dish for reflection of the reference beam in a holographic recording of mental activity. Cranial faults distort the reference beam, disturbing the recall of stored memory. He has applied this hypothesis to other areas of the body, such as the vertebrae having a "bone memory." These hypotheses have been applied on a clinical basis and appear to return disturbed function to normal.*

This discussion on the role of the cranium in health puts into perspective some of the ways disturbance of the cranial primary respiratory mechanism can influence health. It will be observed as our discussion continues that functional problems can develop literally throughout the body as a result of a disturbance in this mechanism. There will be specific sections relating to most of the factors briefly discussed above.

*The holographic hypothesis as applied to body function which Goodheart has presented will be discussed, and references for the complete subject will be given in Volume III of this series.

Etiology of Cranial Faults

There are many ways that disturbances can develop in the cranial primary respiratory mechanism. It is of value to categorize the etiology because it helps organize in the physician's mind the body language indicating the possibility of dysfunction in this area. The primary causes which will be outlined here are (a) trauma, (b) remote structural imbalance, (c) cranial muscle imbalance, (d) habit patterns, and (e) chemical causes.

TRAUMA

Disturbance in the cranial primary respiratory mechanism appears to be most commonly caused by direct or indirect trauma. The trauma often may not be associated with the individual's current health problem because the symptoms sometimes fail to develop until long after the traumatic episode. The symptoms may develop at any time from immediately after the involvement to many years later. The most common types of trauma are (a) birth trauma, (b) forces directed to the skull, (c) indirect trauma, such as a cervical injury with whiplash dynamics, and (d) trauma resulting from dental procedures.

When trauma is part of the history, the physician must make a thorough examination to differentiate conditions such as hemorrhage in the cranium, nerve tissue damage, etc., to which cranial fault thera-

peutics are not applicable. The physician should already be familiar with the diagnosis of headache, head injuries, stupor, and coma. The student is referred to the many texts and papers available on this subject.^{11, 33, 34, 49}

Birth Trauma

It appears that one of the most common causes of cranial faults is birth trauma. Upledger⁶⁴ found a statistically significant positive correlation of difficult birth with dysfunction of the cranial-sacral primary respiratory system. The study was done on school children using the osteopathic concept of examination. Although the full-term skull is very flexible for adaptation to the birth process while still protecting the infant's nervous system, there often appears to be trauma to the skull resulting in cranial faults. Because of major cartilaginous and membranous sections and lack of sutures, the skull molds to navigate the birth canal. Under normal circumstances, the respiration and crying of the child re-expand and activate the skull to its normal function. Whether crying is necessary for the skull's re-expansion is controversial. Some students of the cranial mechanism state that it is absolutely necessary, and failure to do so as a result of the mother's anesthesia impairs normal development.⁴⁵ Others

Chapter 3

Cranial Osteology

Introduction to Cranial Anatomy and Physiology

The general landmarks of the skull are presented first to reacquaint the physician who may have been away from cranial anatomy for some years. Syndesmology is also discussed relative to the sutures. Anatomy and syndesmology are discussed in a manner similar to standard text presentation. This is done to reduce the study time necessary in looking up anatomy in various texts and atlases.^{5, 17, 25, 28, 37} In addition to the standard anatomy of the bones and syndesmology, there is emphasis on the shape of the sutures and also on the type of motion available in the different articulations.^{4, 6, 12, 16, 22, 23, 36}

The bones of the skull have a marvelous design for housing and protecting their associated organs, glands, and structures, and for maintaining the movement necessary for normal function. As previously noted, dynamic function of the skull has just recently received recognition. Because of this, standard texts have not adequately described the anatomy of the sutures. The sutures have a specific design as they change from section to section. Each cranial bone has the ability to move in a predetermined manner. A voluminous amount of material has been written in anatomy texts, but it relates more specifically to the projections, foramina, landmarks, etc., of the bones. This section first presents

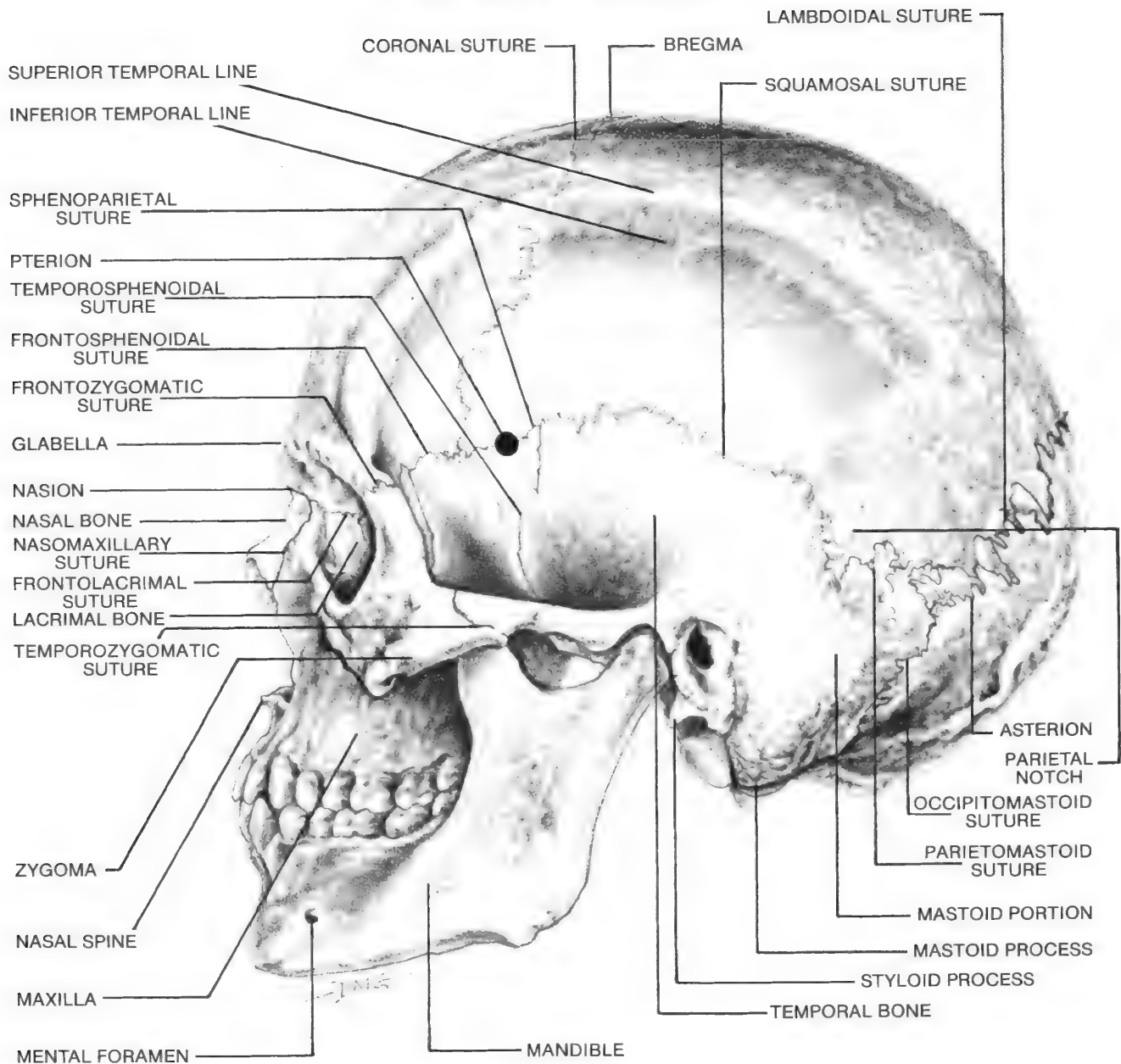
an overall brief description of skull motion and then a general review of each bone's anatomy that is important to the doctor interested in cranial function. The physiologic movement of the bone is then described, and to finish the description there is a discussion of the sutures of the bone, giving the name, type, and motion.

After the description and discussion of each bone, there is a section correlating each bone's movement in total cranial motion with a review of the motion available at each suture. Movement with thoracic respiration will be described, when applicable.

Muscle activity has a significant bearing on cranial function. The muscles can be divided into groups: cranial, facial, masticatory, and postural. Chapter 4 has a description of the cranial and facial muscles. The muscles of mastication are covered very thoroughly in the temporomandibular joint section of this book. Postural muscles are presented in Volume I.

The cranial nerves are described and discussed in Chapter 5, with the possible ways they may be disturbed by cranial primary respiratory dysfunction. Also discussed is the standard method of cranial nerve evaluation.

General Landmarks



3—1. Lateral view.

LATERAL VIEW

Asterion: the region where the lambdoidal, occipitomastoid, and parietomastoid sutures meet.

Pterion: the region where the frontal, parietal, temporal, and superior portions of the greater wing of the sphenoid join. It is a general area rather than an exact location.

Temporal lines: there is a superior and an inferior temporal line. The superior temporal line is the attachment point for the fascia of the temporalis muscle, and the inferior line marks the periphery of

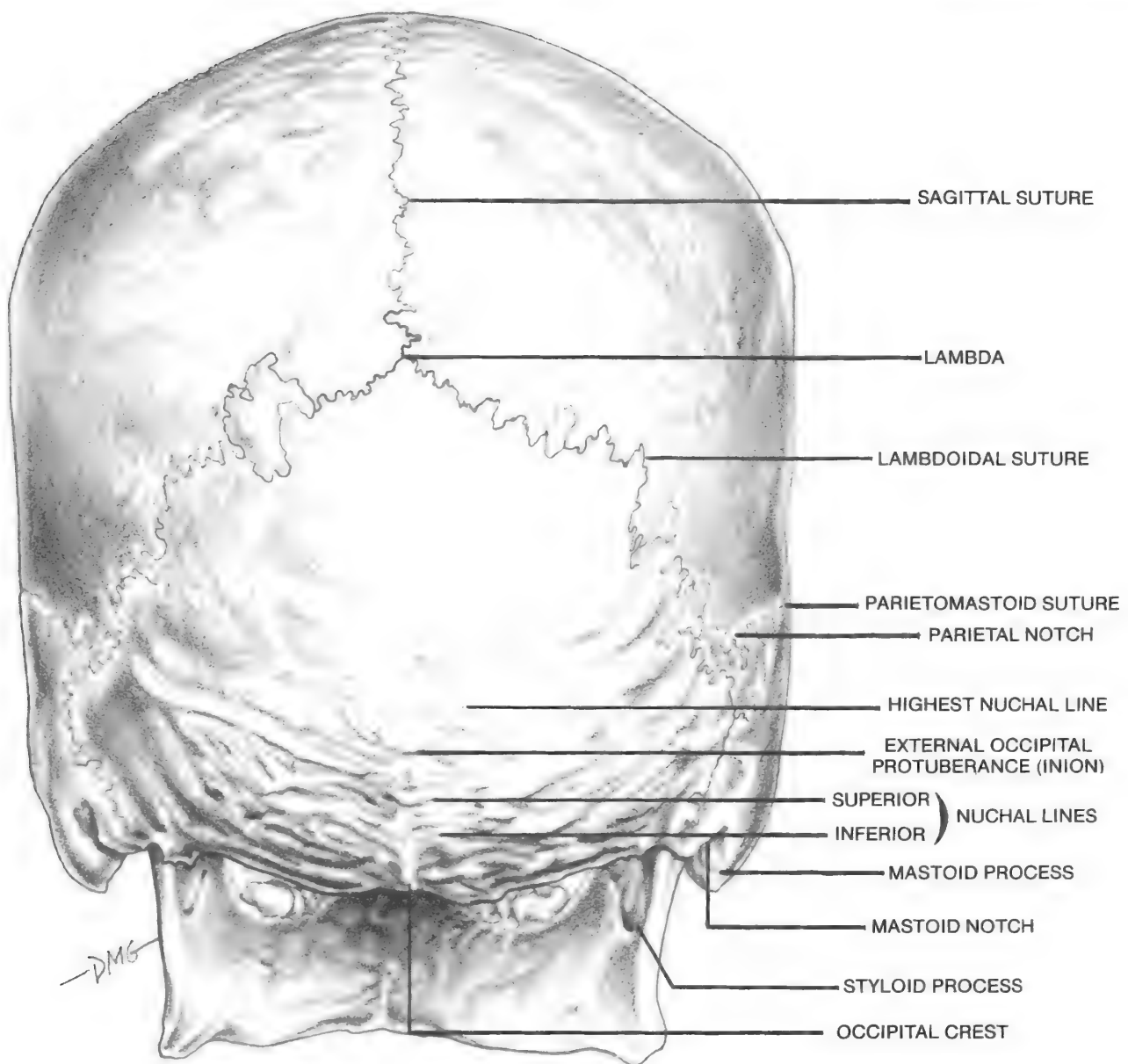
the muscle.

Mastoid angle of parietal bone: articulates with the temporal and occipital bones and is the location of the mastoid fontanelle before ossification.

Parietal notch of temporal bone: this general area often has sutural (wormian) bones.

Mastoid portion: this part of the mastoid is above the general axis of temporal bone rotation.

Mastoid process: this part is below the general axis of temporal bone rotation.



3—2. Posterior view.

POSTERIOR VIEW

Lambda: location where the parietal bones and occipital bone join. Location of the posterior fontanelle before ossification.

Highest nuchal line: a very fine line to which the galea aponeurotica is attached.

Superior nuchal line: arises from the external occipital protuberance and provides an attachment point for the insertion of the upper trapezius, sternocleidomastoid, and splenius capitis.

Inferior nuchal line: immediately inferior to the

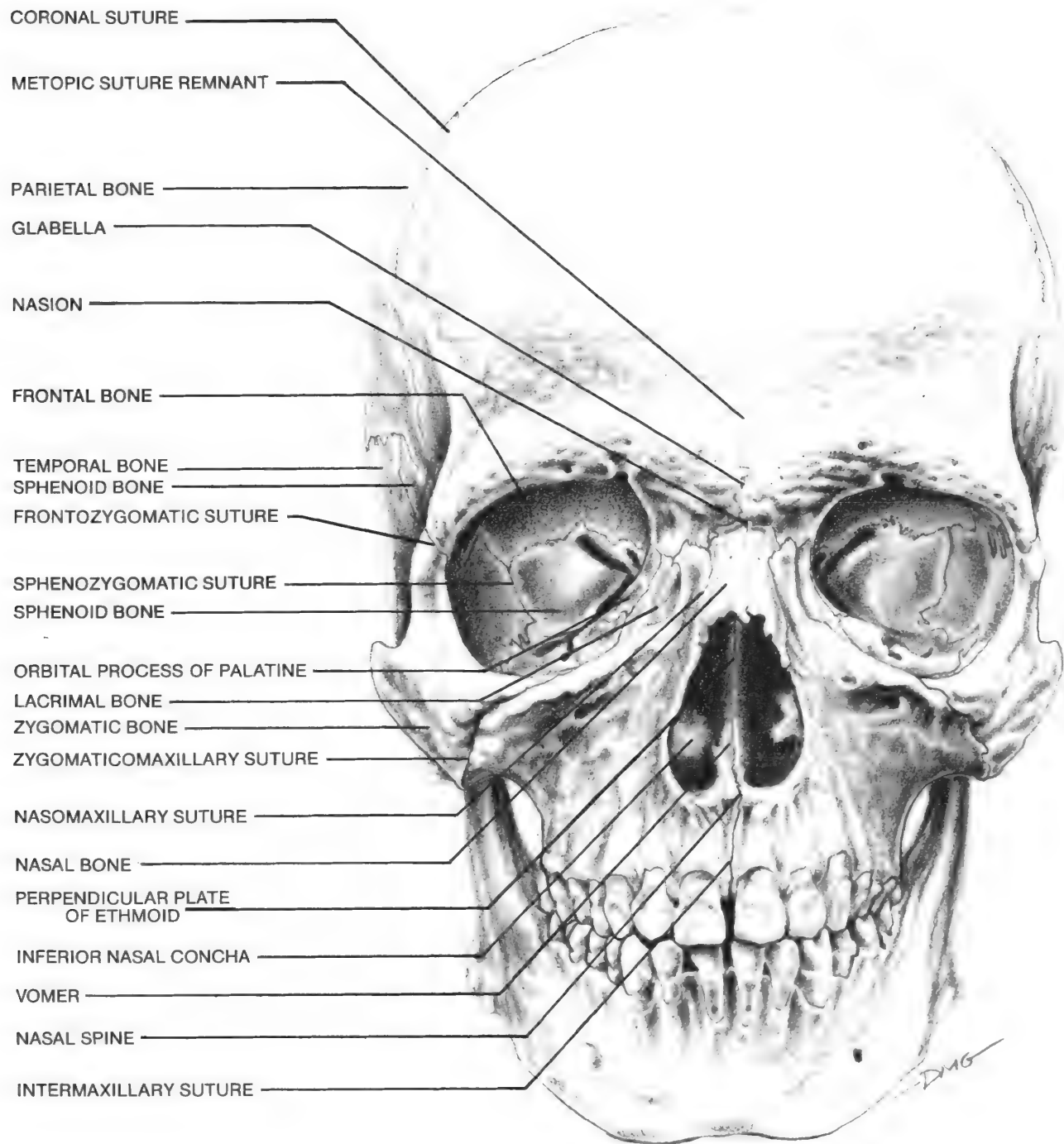
line is the insertion of the rectus capitis posterior major and minor muscles.

Occipital crest: a crest that runs from the external occipital protuberance to the foramen magnum.

Mastoid notch: insertion for the posterior belly of the digastric muscle.

External occipital protuberance: also called the inion.

Cranial Osteology



3—3. Anterior view.

ANTERIOR VIEW

Metopic suture (remnant); at birth, separates the two halves of the frontal bone; begins to ossify at approximately two years of age. Generally there is complete ossification by eight years of age. A remnant or a total suture possibly persists throughout life.

Intermaxillary suture: joins the two maxillae and appears to have movement in cranial and occlusal therapy.³

Seven bones of eye orbit: frontal, zygomatic, maxillary, lacrimal, ethmoid, sphenoid, palatine.

who are actively studying natural birth processes, which are becoming popular today, state that crying immediately after parturition is unnecessary and undesirable.

The normal activity of the infant nursing causes considerable pressure on the hard palate, activating and balancing the cranial mechanism. This action may not be sufficient because of sedation carried over from the mother. The use of a large nipple on a bottle does not require as much activity of the infant's tongue as normal breast feeding.⁴²

There are many ways trauma to the skull can develop at birth. Obviously, when the head is too large for the birth canal, excessive molding must take place which can potentially cause damage. If the sedation used for the mother infiltrates to the child, there is lack of spontaneous respiration and perhaps crying to re-expand the skull. In some instances the trauma to the skull is iatrogenically caused by the improper use of forceps,⁴² and sometimes even from holding the mother's knees together to retard birth until the doctor arrives.

Frymann¹⁹ made a study of 1,250 unselected newborn infants to determine if there was a correlation between cranial dysfunction and the symptomatic pattern. The symptoms of the child were classified regarding "nervous symptoms" which included vomiting, hyperactive peristalsis, tremor, hypertonicity, and irritability. Her study suggested a significant relationship between various forms of cranial-sacral primary respiratory dysfunction and the symptomatology of the child. When disturbance was observed, correction by osteopathic methods improved the child's clinical picture. An interesting observation in this study is that 729 infants were asymptomatic, but the examination revealed structural strain patterns. Presumably, this correlates with cranial imbalance which is present throughout childhood but does not cause symptomatic patterns until possibly the second, third, fourth, or fifth decade of life. It seems reasonable that since the skull is very flexible in youth and becomes less flexible with age, the distortions are tolerated by the body until ossification becomes more complete.

If severe birth trauma is present, yielding dysfunction in the cranial primary respiratory mechanism, the question must be addressed as to whether the child will develop full potential. Dobbing and Smart¹⁷ established that there is an adverse influence on brain development when there is undernutrition during the brain growth-spurt. The brain growth-spurt is a period ranging from near the end of the second human (fetal) trimester to about two years of post-natal age. In another study, Dobbing and Sands¹⁶ demonstrated that the brain has a once-only

opportunity to grow correctly, and it cannot be recovered subsequently. If there is growth retardation during the brain growth-spurt, it is a permanent condition. Among those knowledgeable about the effects of the cranial primary respiratory mechanism, it is widely acknowledged that much cranial disturbance originates from birth trauma. Whether this early dysfunction can influence the individual's potential by affecting the brain growth-spurt is unknown, but it deserves much attention in future research.

It is observed in studying numerous disarticulated skulls that there are variations in the sutural patterns among specimens. As the fontanelles close and the sutures are formed, they are formed in the manner in which the skull can best function in its primary respiratory movement. If the skull is distorted from birth, it is possible that the ossification can develop in such a manner that motion is maintained but varies slightly from the normal. It is also observed in various specimens that accessory (wormian) bones form, usually at specific locations in the skull. Continuing with the hypothesis that the skull ossifies in the best manner for normal motion, it seems reasonable that the wormian bones which appear to form at major areas of stress are for improved motion.

General Trauma

Injury of almost any nature to the head can create cranial faults. The injury may be caused by raising up underneath a kitchen cabinet, striking the head on a car door, or any manner of directly bumping the head.

There are many similarities with the etiology of a vertebral subluxation. The trauma may or may not create a problem, depending on the sum of the circumstances. If there is balance within the body, particularly the stomatognathic system, there will probably be a self-correction. The body is a self-correcting, self-maintaining mechanism. The physician needs to step in only when the body is incapable of making its own corrections. The symptomatic pattern from a cranial fault may not develop until many days, weeks, months, or longer after the actual fault has developed. This is similar to the often observed delay of symptoms from a vertebral subluxation.

"Whiplash" Dynamics

The hyperflexion-hyperextension cervical trauma often seen in automobile accidents and loosely termed "whiplash" is a potent force which may directly or indirectly cause cranial faults.³⁸ The insertion of muscles — such as the sternocleidomastoid and upper trapezius — into the cranium can

Introduction to Cranial Function

cause direct pull on the skull when the muscle is severely stretched, creating a cranial fault. On the other hand, the actual trauma may not directly create a cranial fault; it can do so indirectly. If muscles attaching to the skull are injured, causing an imbalanced pull into the cranium with daily activities, cranial faults may develop secondarily and at a later time. Returning the muscles to normal function may or may not correct the cranial dysfunction. The ideal therapeutic approach gives attention to the muscles involved in the trauma, and also examines for and makes correction of any cranial dysfunction.

The muscles attaching to the hyoid, and also facial muscles, may ultimately create cranial faults if injured. These will be discussed later under their respective subjects.

Sometimes cranial faults can be created iatrogenically when the doctor is treating some other area. This most frequently develops as a result of traction, which is applied by various methods for cervical injuries. The traction halter applied generally to the occiput and mandible can place pressures into the cranium in such a way that its function is disturbed. If the body is not capable of restoring normal function, a cranial fault results. An individual may have cervical spine discomfort as a result of an auto accident which produced "whiplash" dynamics to the neck. The injury may have created cranial faults and consequent spinal accessory nerve involvement, affecting the sternocleidomastoid and upper trapezius. A common type of treatment administered for this condition is cervical traction, which may in effect perpetuate the cranial disturbance or even make it worse.

Spinal manipulation can be the source of iatrogenic cranial faults. When an upper cervical or occipital subluxation or fixation is corrected, the doctor may contact the skull in such a manner that the cranial bones are jammed or distorted, creating the cranial fault.

Dental

Subdivisions must be made under the classification of dental as etiology for cranial faults. First is the individual who has a malocclusion for any of many reasons. This may produce cranial faults by imbalanced forces being placed into the skull every time the individual chews or bites down. In this case it is often necessary for dental procedures to be performed along with the cranial corrections for lasting results. Second, cranial dysfunction can result as an iatrogenic factor following various dental procedures. Henningsen,^{31, 32} a dentist knowledgeable about the cranial-sacral primary respiratory system, cautions his profession that "Techniques should be completed

with the greatest of care." Furthermore, "... the use of elevators, forceps, and mouth props ... can be powerful fulcra." Holding the mouth open for prolonged dental procedures can cause cranial faults, as can the extraction of teeth. A crown can be responsible for cranial dysfunction. This can develop as a result of pressures used when seating a crown, or failure to correctly equilibrate the crown, causing an occlusal problem. Whenever there is an occlusal problem, strains are placed into the cranial primary respiratory mechanism every time the individual chews, swallows, or bites down forcefully.^{37, 44}

STRUCTURAL IMBALANCE

By observing function as remote from the skull as possible, an example can be made of how structural disturbance almost anywhere in the body can create cranial dysfunction. The foot supplies this example. Normally, when an individual runs, walks, and generally functions in an upright position, muscular contraction is rhythmic and synchronous with the body's needs. This organization, of course, comes from proprioceptive signaling, and ultimate muscular coordination results. In the presence of foot subluxations, improper signaling develops, causing a lack of proper coordination between muscles. The muscles of the upper sacrospinalis, sternocleidomastoid, upper trapezius, hyoid muscles, etc., can contract at inappropriate times, pulling on the cranium in such a manner as to disturb the cranial primary respiratory mechanism. This is accentuated by improper muscle function influencing the pelvis, including the sacrum, which is organized in the primary respiratory mechanism.

The movement of the skull, designated as the cranial primary respiratory mechanism, is directly correlated with a similar movement of the pelvis called the pelvic primary respiratory mechanism. The primary activity is that of the sacrum, which is organized with skull activity by way of the dura mater. The innominate bones also have a movement correlating with cranial activity. If an individual has a persistent pelvic disturbance, usually a category I, there may be a corresponding cranial disturbance. Correction of the cranium probably will not hold until the pelvic dysfunction is corrected.

The pelvic disturbance can be from direct trauma, remote structural dysfunction, or imbalance of the muscles supporting pelvic activity, such as the sartorius, gracilis, hamstrings, abdominal muscles, gluteus maximus-medius-minimus, etc. (Pelvic categories are discussed in Volume I and later in this text.)

The effect of remote structural problems creating disturbance in the primary respiratory mechanism is

observed clinically by first correcting all pelvic and cranial dysfunction, and then having the patient walk or run. Disturbance of a remote nature will cause the primary respiratory dysfunction to return immediately. After the foot or other responsible structure is corrected, the corrections made to the primary respiratory mechanism after the patient runs, walks, jumps, etc., should hold.

CRANIAL MUSCLES

Influence of the cranial muscles on the skull can be observed in a study by Washburn,⁶⁹ who removed a temporal muscle from day-old rats. The imbalance of muscular activity caused the architecture of the cranium to change, including the sutural design. The influence on the skull is more significant from imbalance of the muscles of mastication. These very powerful muscles are active in skull movement and, when out of balance, can create or perpetuate cranial faults. Correction may simply be attention to the muscles with the usual applied kinesiology procedures; it may require dental procedures for equilibration. The intrinsic muscles of the skull (primarily facial muscles) can also influence cranial activity. Applied kinesiology evaluation with muscle stretch reaction, Golgi tendon organ, and neuromuscular spindle cell can find these imbalances and correct them, returning cranial activity to normal.

HABIT PATTERNS

There are many habit patterns that can create or perpetuate cranial faults. The young student with a very flexible skull may habitually sit at a desk to read, propping his mandible on one hand. This puts strain

into the temporal bone, which is a key bone in cranial motion. Adults often read in bed, lying on their sides and propping their heads up with an arm. This throws strain into the skull in a similar manner. Pipe-chewing, or holding other foreign objects in the mouth in consistent patterns, can distort the cranium and create a cranial fault or perpetuate it. Even excessively tight headgear — such as welding helmets, hats, etc. — can influence the mechanism.

Sometimes work habits may be the culprit. Sheetrock installers often use their heads to hold sheets for ceiling installation. They frequently develop headaches and cervical problems which may be due to trauma in the neck or in the cranium.

An occupational distortion — such as that developed by hod carriers, porters, the old-time railroad engineer, etc. — can cause strain to the entire myofascial system, which places distortion into the skull.⁶

CHEMICAL

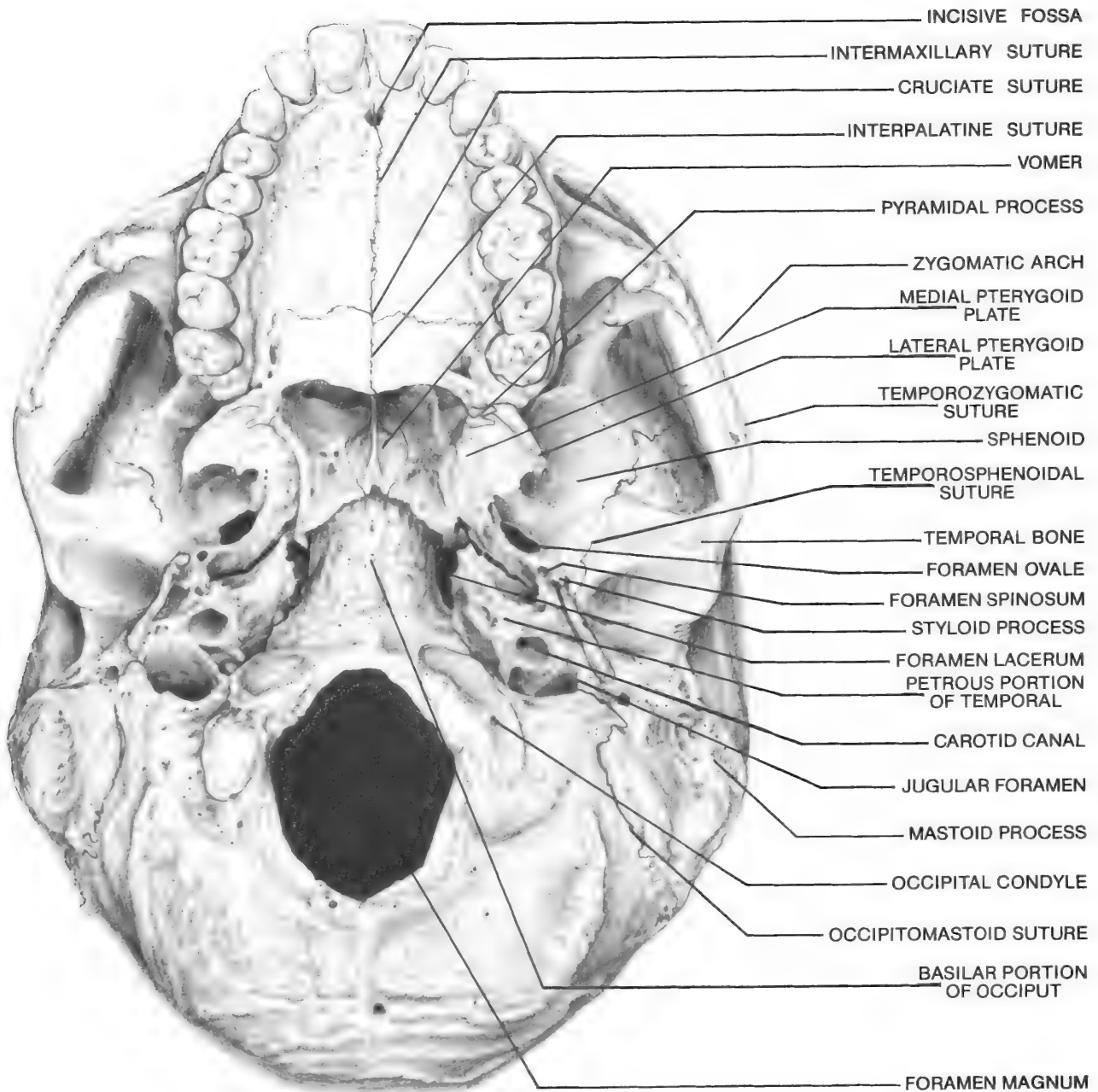
The mechanism of chemical factors creating cranial faults is not well understood and may be difficult to accept at first. Clinical evidence of chemicals causing cranial faults is often observed. A cranial fault may be corrected on a patient, and then oral administration of a particular substance, entering the nervous system by way of the gustatory mechanism, may re-create the evidence of the previously corrected cranial fault; an antagonistic chemical substance immediately removes the evidence of the fault when administered in the same manner.

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3—4. Inferior view.

INFERIOR VIEW

External occipital protuberance: also called the inion.

Mastoid notch: insertion for posterior belly of the digastric muscle.

Mastoid process: below the general axis of rotation of the temporal bone; moves posteriorly and medially on sphenobasilar flexion (inspiration).

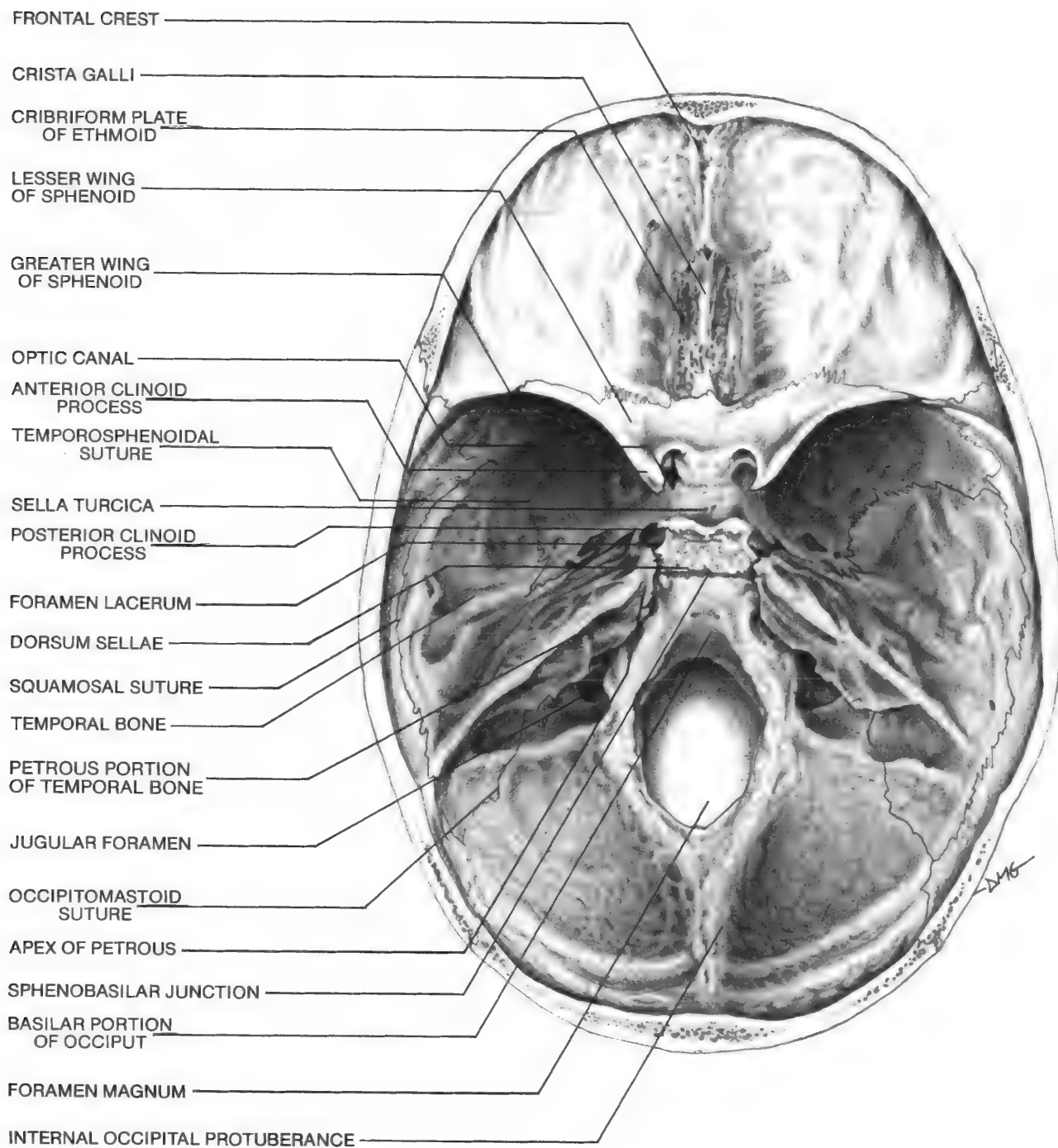
Styloid process: origin of stylohyoid muscle.

Pterygoid process: note the lateral and medial

pterygoid plates with the intervening pyramidal process of the palatine bone.

Cruciate suture: the cruciate suture is a cross-like suture composed of the intermaxillary, interpalatine, and palatomaxillary sutures. Pressure applied at this area can direct force through the palatine bone to the pterygoid processes and through the vomer, thus influencing the sphenoid.

Cranial Osteology



3—5. Cranial floor.

Cranial Primary Respiration

When discussing cranial function, remember that there are two types of respiratory correlation. The first is the cranial primary respiration originally described by Sutherland. This respiration is independent of the diaphragmatic respiration of breathing. In this text, cranial primary respiration is always referred to in that way. Diaphragmatic respiration is simply called respiration, breathing, or thoracic respiration.

Different motions of the skull are enhanced with different phases and types of breathing. There is a specific respiratory correlation with most cranial faults in applied kinesiology. These phases of respiration appear to enhance specific cranial movements. The basic motion of the skull is outlined here, and then discussed more thoroughly with each bone of the cranium. A summary of skull motion and its evaluation concludes the discussion.

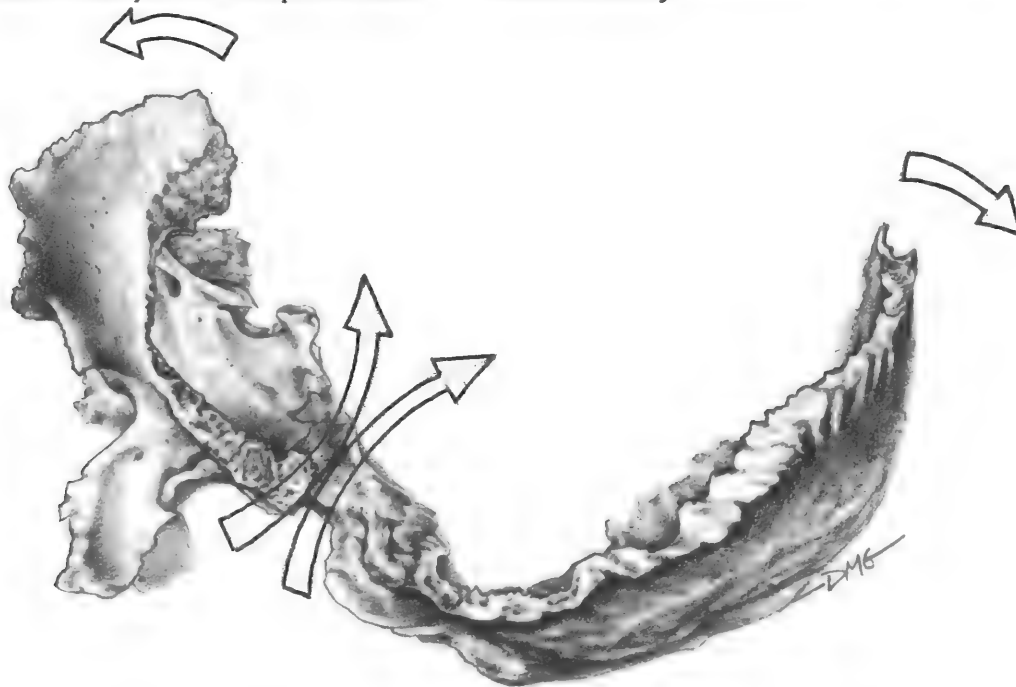
As we begin to consider the motion of the skull more thoroughly, it should be noted that there is considerable disagreement between various schools of thought about precisely how each cranial bone moves. The motion accepted in applied kinesiology is basically that which Sutherland³⁶ described. This motion is supported by muscle testing procedures as discussed on page 87.

Motion of the skull can best be understood if motion of the sphenoid and occipital bones is first considered. These bones join at the sphenobasilar

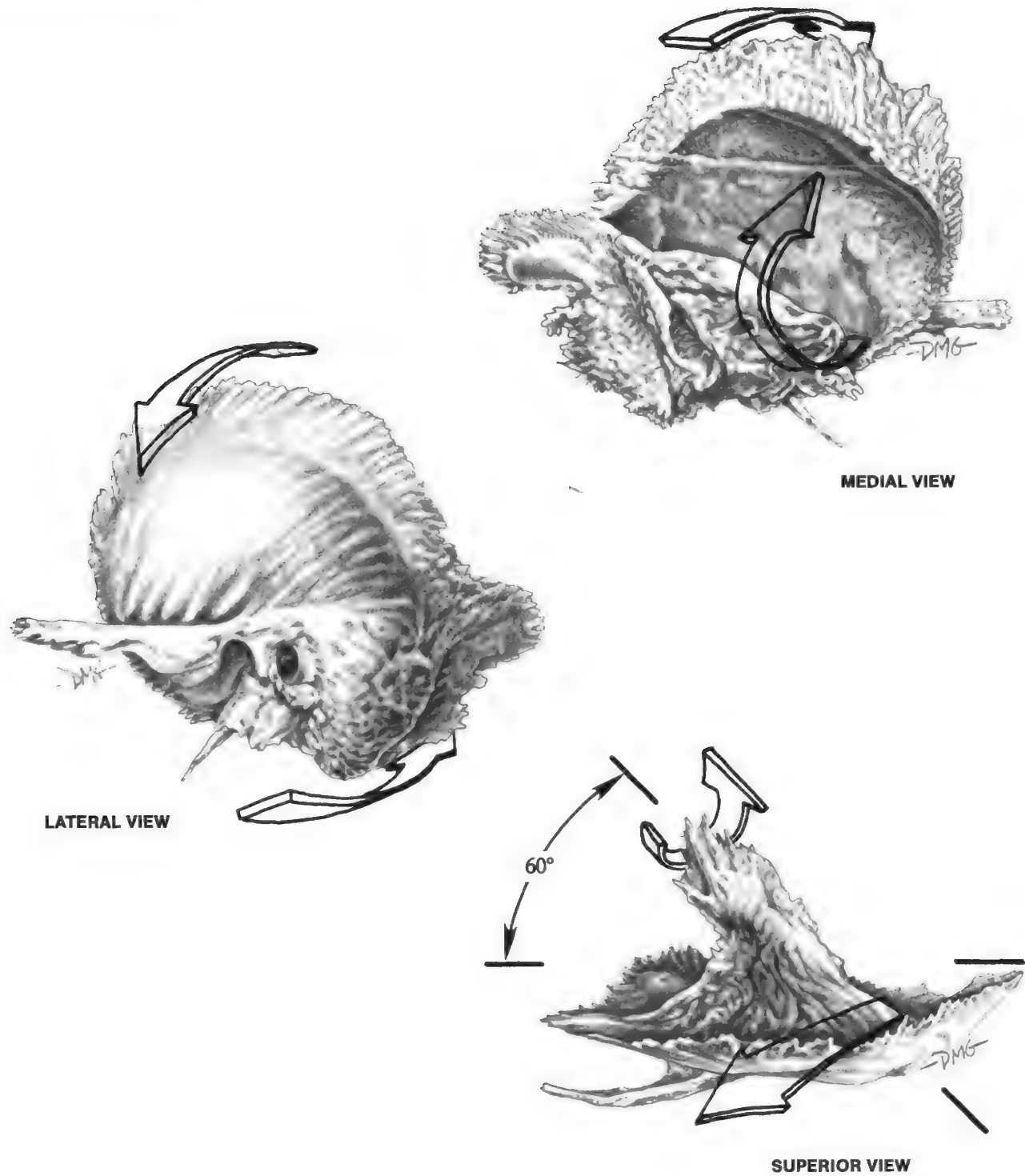
junction. When the terms "inspiration" and "expiration" are used, they refer to both thoracic respiration and movement of the cranial primary respiratory mechanism. The terms "flexion" and "extension" refer to activity at the sphenobasilar junction. Flexion of the sphenobasilar junction is synonymous with inspiration, and extension of the sphenobasilar junction is synonymous with expiration.

On inspiration the sphenobasilar junction flexes, which means it moves superiorly. At the same time, the superior portion of the squama of the occipital bone moves posteriorly and inferiorly, while the superior lateral aspect of the greater wing of the sphenoid moves anteriorly and the floor portion of the greater wing moves posteriorly.

The sphenoid rotates through an approximate axis anterior to and slightly below the floor of the sella turcica. This point is in alignment with the sphenosquamous pivot, usually located slightly above the level of the zygomatic process. The posterior floor of the greater wing of the sphenoid moves posteriorly and superiorly, while the superior lateral margin of the greater wing moves anteriorly. The L-shaped suture on the superior and lateral borders of the orbital surface moves inferiorly. The horizontal portion bordering the superior orbital suture moves slightly anteriorly. The pterygoid process moves posteriorly, and the anterior portion of the body moves inferiorly.



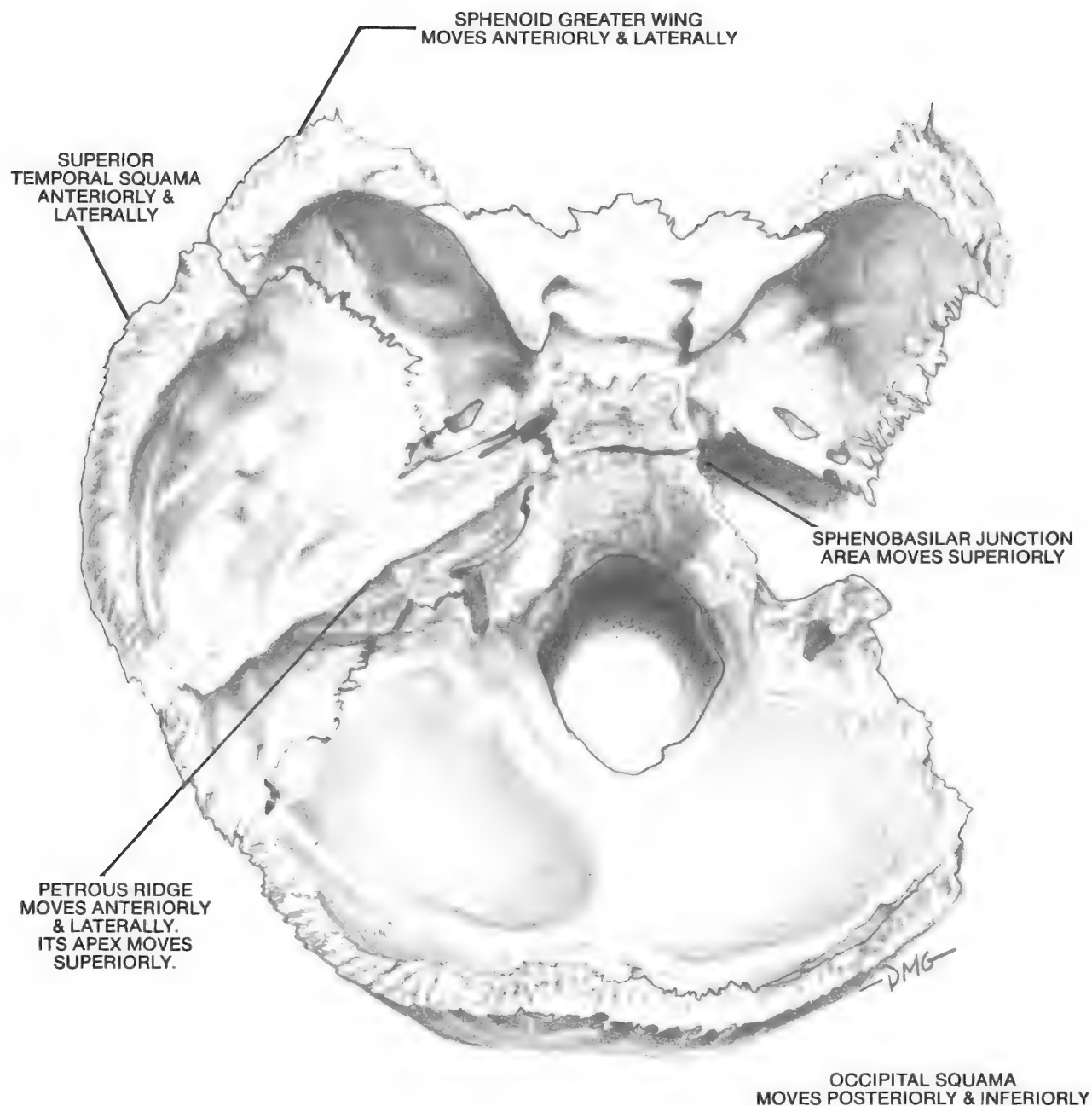
3—6. The arrows indicate the general motion of the sphenoid and occipital bones on sphenobasilar flexion (inspiration).



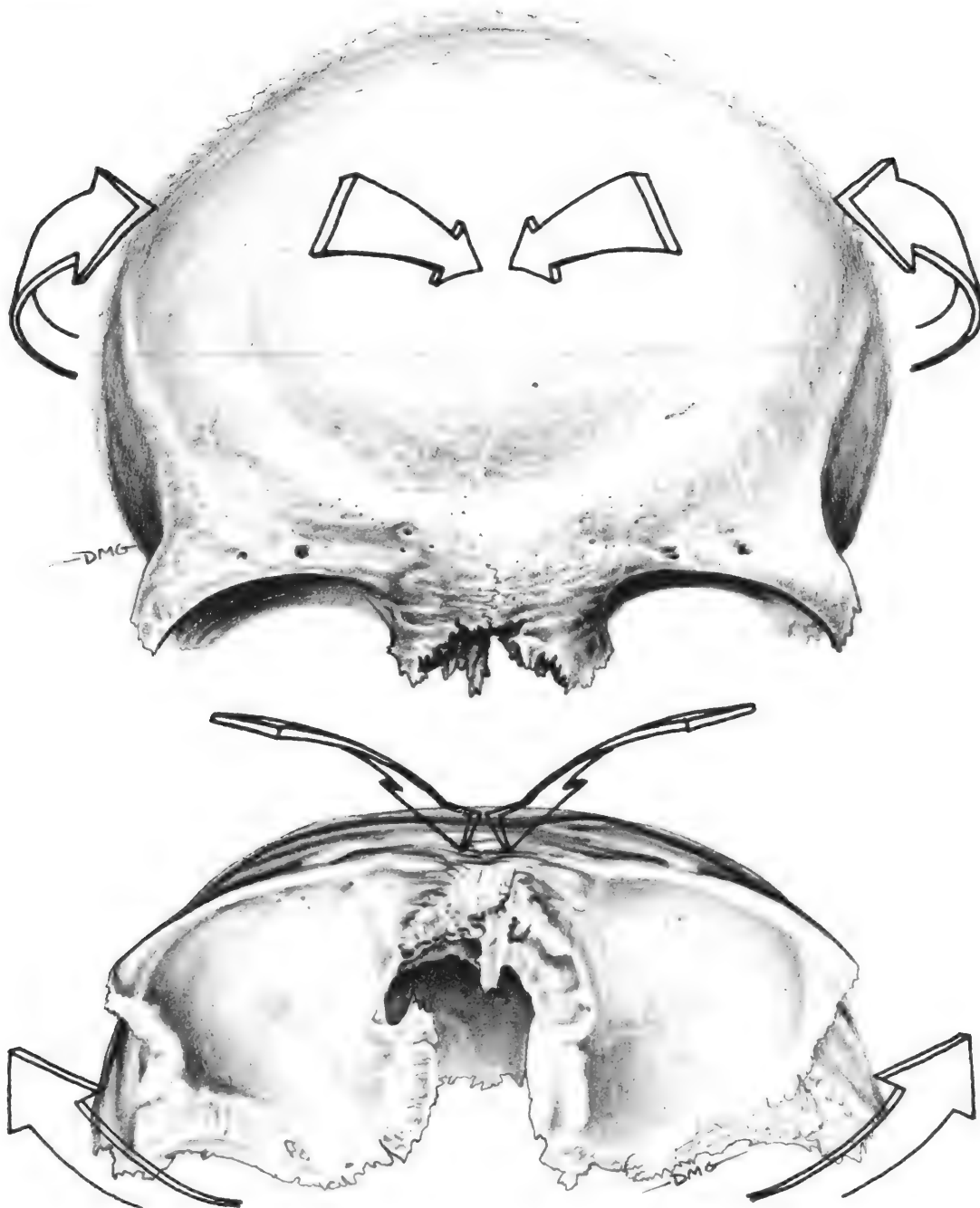
3—7. Temporal movement on sphenobasilar flexion (inspiration).

The temporal bone rotates around an approximate axis through the petrous portion. This causes the squamous portion of the temporal bone to rotate externally, or bulge. There is also anterior motion of the superior aspect of the squamous portion. The

anterior portion of the squama — inferior to the zygomatic process — moves posteriorly and medially. The mastoid portion of the temporal bone moves anteriorly and laterally, while the mastoid process moves posteriorly and medially.



3—8. Motion on sphenobasilar flexion (inspiration). This combined movement of the sphenoid, occipital, and temporal bones requires specific activity at various sutures. This will be explained with the individual consideration of each bone and in the final description of motion in this chapter.



3—9. Arrows indicate the direction of movement of the frontal bone on sphenobasilar flexion (inspiration).

The frontal bone has a two-dimensional movement, anterior and posterior tilting, and a spreading of the internal concave surface of the squama which depends upon the flexibility of living bone. The spreading of the squama can best be visualized if the frontal is considered as two bones, as if the metopic suture were persistent. This, in fact, is the way terminology has developed in cranial evaluation and

treatment systems. Rotation of the frontal bone about the vertical axis during sphenobasilar flexion is that of external rotation of the lateral posterior aspect of the squama, and internal rotation at the metopic suture area. The articulation of the sphenoid and frontal bones moves slightly forward, while the superior aspect of the coronal suture moves slightly posteriorly and inferiorly, depressing the bregma.

Skull Syndesmology

Introduction

There will be considerably more discussion about the articulations of the skull in this text than in the classic anatomy texts. The typical anatomy text states that the skull does not have movement; its primary purpose is to house and protect the structures contained within it. In fact, Gray's *Anatomy* (British edition)³⁷ states, regarding what is considered as the progression to "rigid synostosis," that "... sutural fusion does not even commence until the late twenties, proceeding slowly thereafter; yet it is clearly necessary that sutures should cease to function as mobile joints as rapidly as possible after birth." With this attitude toward skull movement, the authors' lack of description of the changes of sutural design at specific locations of the articulation is understandable.

The specific design of each suture will be considered in the section on each bone of the skull. It will be noted that the overall design of the sutures is uniform from individual to individual; however, there are unique variations from skull to skull, just as there are differing characteristics from face to face.

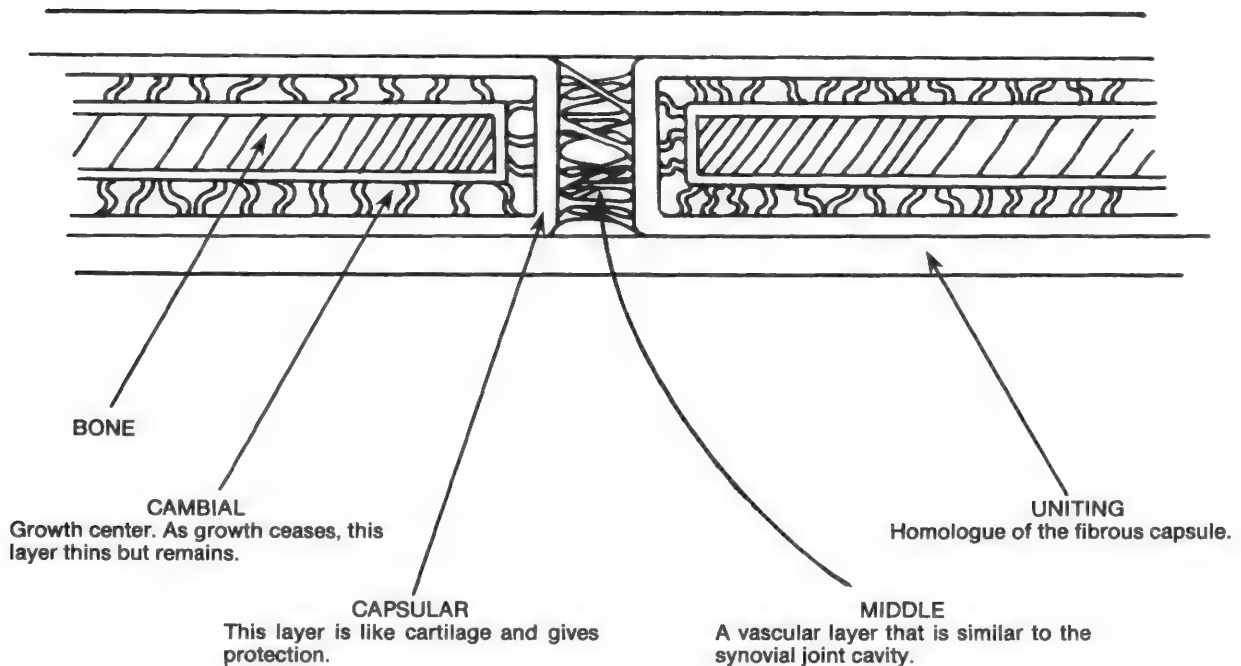
Close study of disarticulated skulls indicates another reason for slight variations from skull to skull. The base of the skull develops in cartilage, while the cranial vault develops in membrane. At the time of birth, a considerable amount of skull motion is necessary for yielding during the birth process. As the skull ossifies and finally develops its adult sutures, the living motion continues. It appears that the ossification process takes place in a manner which allows optimum skull motion. If there has been trauma at birth or other trauma causing lack of symmetry and restricted motion, the skull appears to ossify in an unusual manner, with slight changes in the formation of the articulations. Sutural (wormian) bones may develop as a mechanism to enhance skull motion when it is resisted along normal sutural lines. Testing the hypothesis that sutural bones develop to aid skull motion requires histologic study to determine if there was an accessory ossification center causing the sutural bone to form. This has not been done; however, the hypothesis that sutural bones aid motion in restricted skulls seems reasonable. Sutural

bones are usually found around the occipitomastoid suture, occipitoparietal suture, and other areas where it is recognized that there is often stress in the cranial primary respiratory mechanism. This seems reasonable, especially in light of investigations such as Giblin and Alley's,¹¹ described on page 15. Other studies^{26, 31} also indicate the adjustment of suture to need and displacement of sutural position.

A study was done by Washburn³⁸ where the temporal muscle was removed from newborn rats. This changed the bone growth. Where the loss of muscle decreased bone growth, sutures were simpler than normal. Where growth was prolonged, the sutures became more complex. This seems to indicate that the morphology of the suture develops for the need at that articulation.

Clinical observation does not correlate with the thought that "... it is clearly necessary that sutures should cease to function as mobile joints as rapidly as possible after birth." A physician knowledgeable about the applied kinesiology approach to the cranial primary respiratory mechanism continually observes that people with chronic health problems often have rigid skulls which tend to resist treatment. People who have been in good health during their life-span have more supple, "loose" skulls, whether or not cranial faults are currently present. An individual may be an octogenarian who has been in excellent health throughout life; then he is involved in an auto accident which creates cranial faults. In this case, the physician will nearly always observe that the skull is supple and easily corrected. On the other hand, an individual in the fourth decade of life may have had poor health his entire lifetime and have a very rigid skull with which it is difficult to work. The plastic nature of the skull observed during examination and treatment gives some information regarding the prognosis of the case. This is also observed by the number of mobilization efforts the physician must make before correction is obtained. In the rigid skull, additional numbers of therapeutic pressures must be used on each treatment, and often more treatments are required.

3—10. Suture histology. This schematic representation of a suture, after Pritchard and co-workers,²⁹ gives the homologue of the various layers with the structures in a diarthrodial articulation.



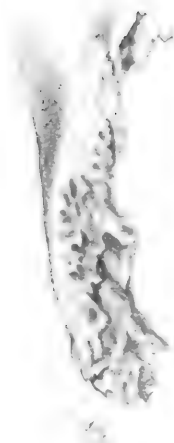
Types of Sutures in the Skull

The articulation known as a suture is unique in the body; it is found only in the skull. There are five layers — cambial, capsular, middle, capsular, and cambial. The articulation is covered by the uniting layer, which is a continuation of the periosteum. The cambial layer is the growth center of osteogenic cells; as growth ceases, this layer thins but does not disappear. The capsular layer is a continuation of the periosteum and appears to be the homologue of the cartilaginous epiphysis of diarthrodial articulations.

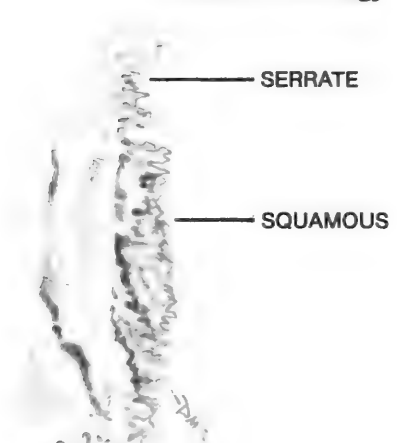
The middle layer, with its weak fiber bundles running in all directions and its sinusoidal blood vessels, appears to be analogous to a synovial joint cavity. The uniting layer is the strongest bond of union between the bones and is evidently the homologue of the fibrous capsule of diarthrodial articulations. Pritchard et al.²⁹ did the histologic investigation for the above conclusions and stated: "Considered as articulations, the sutures possess the means for resisting gross separation of the bones, while at the same time permitting slight relative movement."



3—11. Dentate



3—12. Serrate (view into suture).



3—13. Limbus

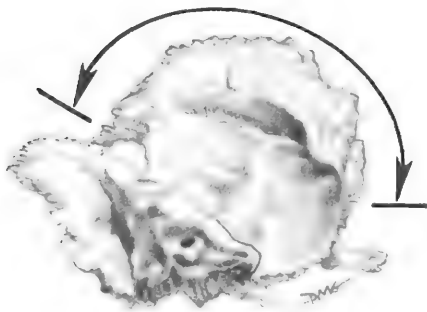
If the bones of the skull are connected with a series of projections and interdigitations, the suture is called a *true suture*. There are three types of true sutures: (1) dentate, (2) serrate, and (3) limbus.

The *dentate suture* derives its name from the tooth-like processes which project into the adjacent bone. This provides a very solid connection. Sometimes the distal aspect of the projection is enlarged, thus increasing the suture's locking capability and resisting gross separation. A primary example is the sagittal suture between the parietals.

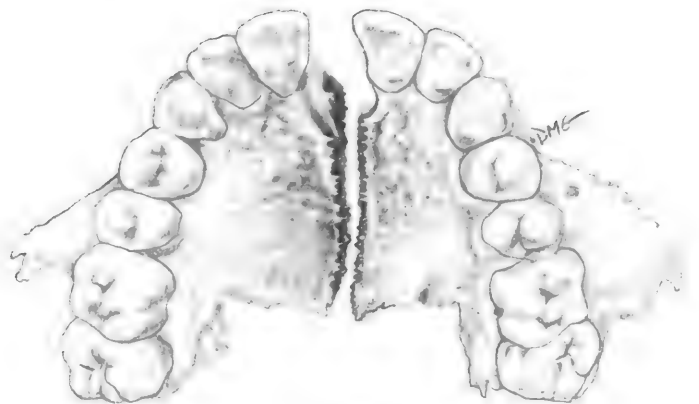
The *serrate suture* is characterized by a saw-

tooth edge interdigitating with the adjacent bone. This suture has an expansion capability at the articulation, as well as good interlocking of the articulation. An example is the temporosphenoidal suture inferior to the level of the zygomatic arch.

The *limbus suture* is serrated, having the same characteristics as the serrate suture. It is also beveled, having some of the characteristics of a squamous suture described later. Examples generally given are portions of the temporosphenoidal and coronal sutures; however, there is significant serration in these sutures.



3—14. Squamous

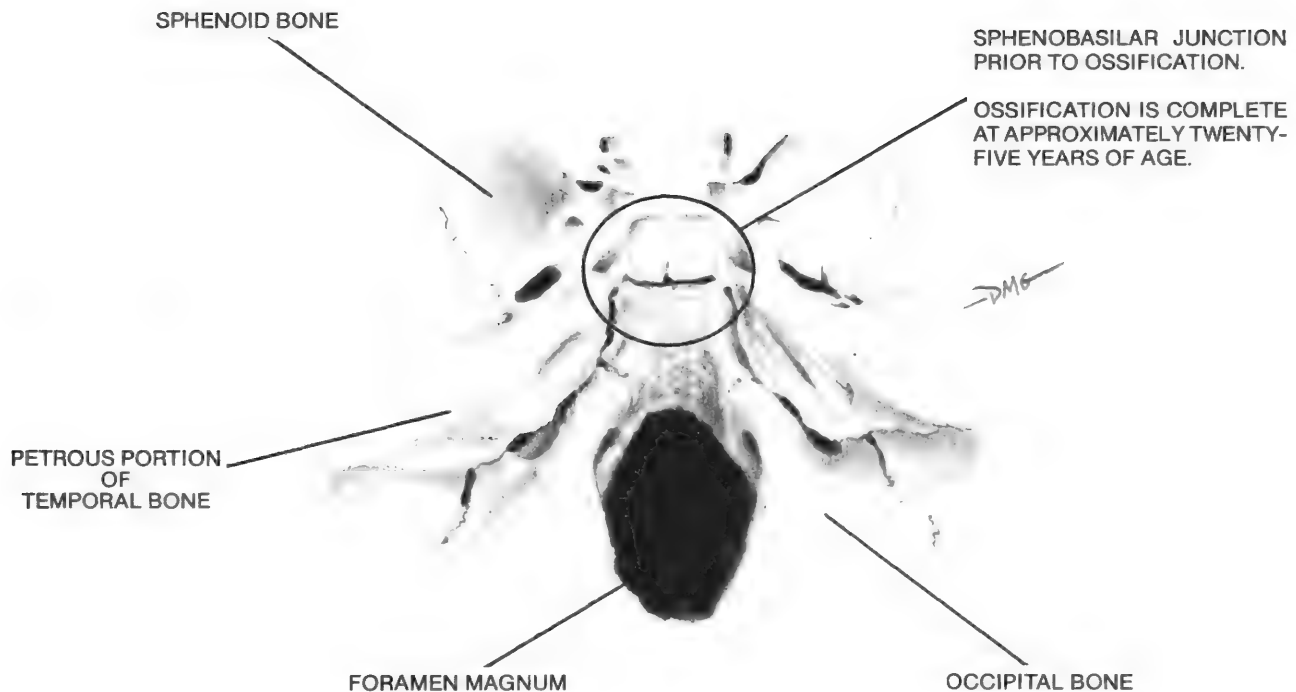


3—15. Plane

Sutures formed by overlapping or a butting of roughened surfaces are termed *false sutures*, of which there are two types: the squamous and the plane (harmonious).

In the *squamous suture* there is an overlapping of the bones by beveled margins. The bevel is termed either an internal or an external bevel. An internal bevel is one which faces toward the internal aspect of the skull, and the internal aspects of the bone have

been sacrificed for the bevel. An external bevel faces the external aspect of the skull, and the external table of bone has been sacrificed. An example of a squamous suture is the squamosal suture between the temporal and parietal bones. A *plane suture* is the simple butting together of two bones. Usually their surfaces are rough, giving the articulation some strength. Examples are the intermaxillary and interpalatine sutures.



3—16. Illustration of the sphenobasilar junction of a dry, defatted skull approximately eighteen years of age.

A *schindylesis* is a suture in which a thin plate of bone fits into a groove of a neighboring bone, such as in the junction between the vomer and the rostrum of the sphenoid bone.

A *gomphosis* is a peg-in-a-socket type of articulation illustrated only by a tooth in the alveolar socket.

Amphiarthrodial articulations are slightly movable and are characterized by a cartilage interposed between the two bones. The two types are *synchondrosis* and *symphysis*. A *synchondrosis* is a cartilaginous articulation which ossifies before or in early adult life. An example is the sphenobasilar junction, which ossifies to cancellous bone at approximately twenty-five years of age. *Symphysis* is an articulation which is united by a fibrocartilage disc. An example is the symphysis pubis of the pelvis.

Limited Motion of Joints

It must be re-emphasized that to appreciate the limited motion of the articulations being discussed, it is necessary to consider the articulations *in vivo*. Sutures such as dentate and serrate are extremely strong and are almost or completely inseparable in

dry, defatted skulls. The projections of the dentate or serrate suture may even have an enlargement at the distal aspect of the projection, thus giving an interlocking factor to the structure. To disarticulate a dry skull, it is necessary to soak it in an attempt to regain some of the plastic nature it had in life. Upon trying to re-articulate a disarticulated skull, it can be easily observed that the projections of the serrate and dentate sutures have changed shape so that they no longer fit together. This in itself should give the investigator an understanding of the flexibility of living bone. Studies such as those by Stowe et al.³⁴ (see page 12) give further evidence of the living bone's flexibility.

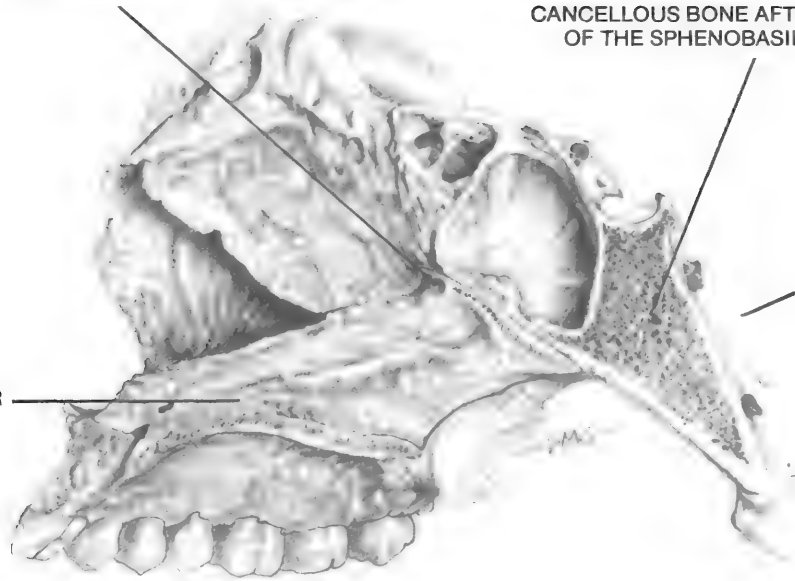
Classification of major joints of the body often seems confusing when comparing text to text. Gray's *Anatomy* (British edition)³⁷ classifies the sacroiliac articulation as a *synchondrosis* which, according to their definition, is "... a temporary form of joint, for the cartilage is converted into bone before adult life." This is disputed by Gray's *Anatomy* (American edition)¹⁷ and by the study of Frigerio et al.⁸

SCHINDYLESIS BETWEEN THE VOMER BONE AND
ROSTRUM OF THE SPHENOID BONE

CANCELLOUS BONE AFTER OSSIFICATION
OF THE SPHENOBASILAR JUNCTION

SPHENOBASILAR
JUNCTION

VOMER



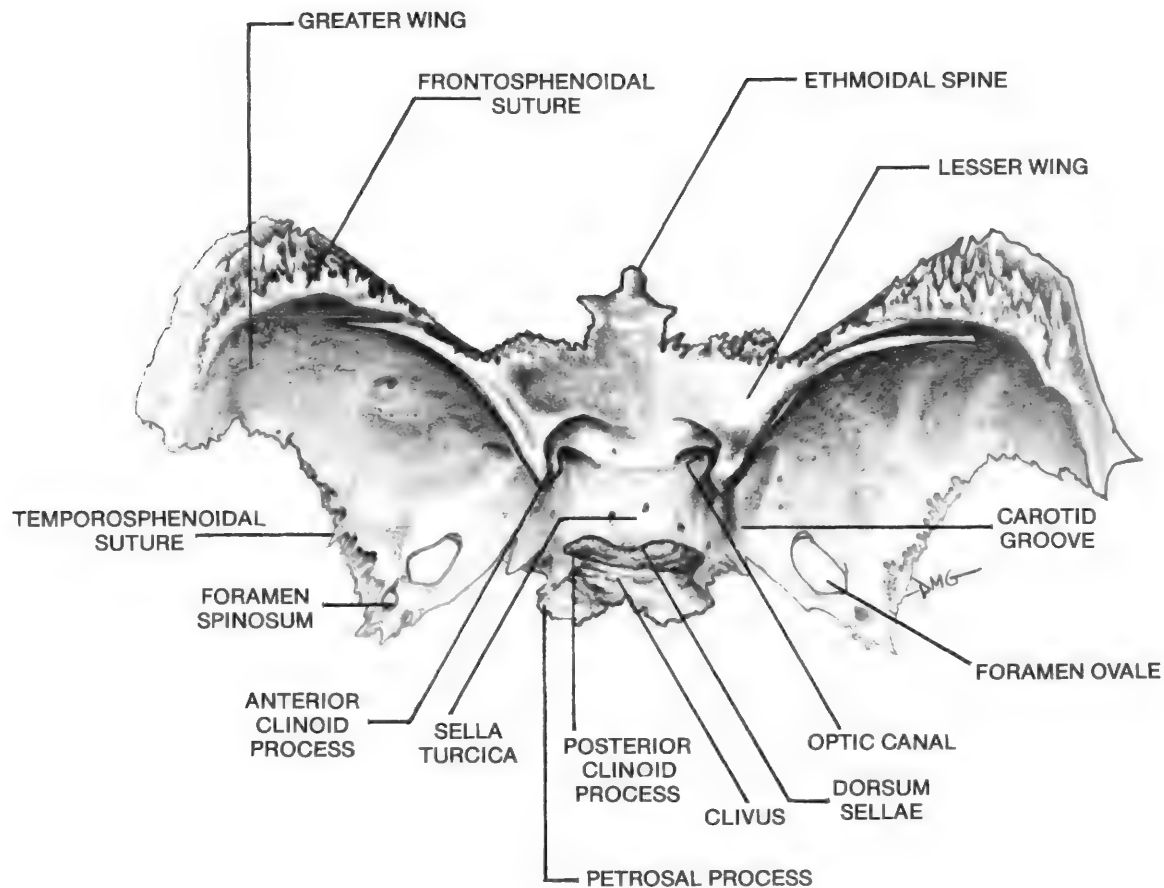
3—17. *Sagittal view of sphenobasilar junction of an adult.*

Sphenoid Bone

The sphenoid is the key bone in cranial movement, playing a primary role in joining the bones of the cranium and face. It articulates with twelve bones — the occipital, frontal, ethmoid, and vomer, and bilaterally with the temporal, zygomatic, parietal, and palatine — and it may have articulation with the tuberosity of the maxilla. The sphenoid is divided into the body, two greater wings and two lesser wings projecting from the sides of the body, and two pterygoid processes which project from its inferior surface.

The body contains the sphenoidal sinuses. Arising from the anterior superior surface of the body is the ethmoidal spine, which articulates with the cribriform plate of the ethmoid. The superior posterior portion of the body is hollowed out and is called the sella turcica; this area holds the pituitary gland. Anterior to the sella turcica are the anterior clinoid processes, while posterior are the posterior clinoid processes. The tentorium cerebelli is attached to the posterior clinoid processes. Just anterior to the sella turcica is the optic groove on which the optic chiasma lies. The groove leads bilaterally to the optic foramen, which channels the optic nerve and ophthalmic artery into the orbital cavity. The posterior

boundary of the sella turcica is the dorsum sellae, which supports the posterior clinoid processes. The notch for the passage of the abducent nerve (cranial VI) for the lateral rectus muscle is bilateral to the dorsum sellae. Inferior to the notch is the petrosal process, which articulates with the apex of the petrous portion of the temporal bone and forms the medial boundary of the foramen lacerum. The posterior aspect of the body slopes sharply inferiorly to its junction with the basilar portion of the occipital bone. The surface is known as the clivus and supports the upper part of the pons. The posterior inferior surface of the body slopes downward anteriorly and joins with the basilar portion of the occipital bone to form the sphenobasilar junction. The sphenoidal crest and concha arise from the anterior portion of the body, and the anterior surface forms the posterior wall of the nasal cavity. The inferior surface also contributes to the posterior nasal wall. In its center line is a continuation of the sphenoidal crest which is called the sphenoidal rostrum. It articulates into the deep furrow between the two alae of the vomer bone. In the anterior region, the sphenoid articulates with the ethmoid and palatine bones and with the orbital plate of the frontal bone.



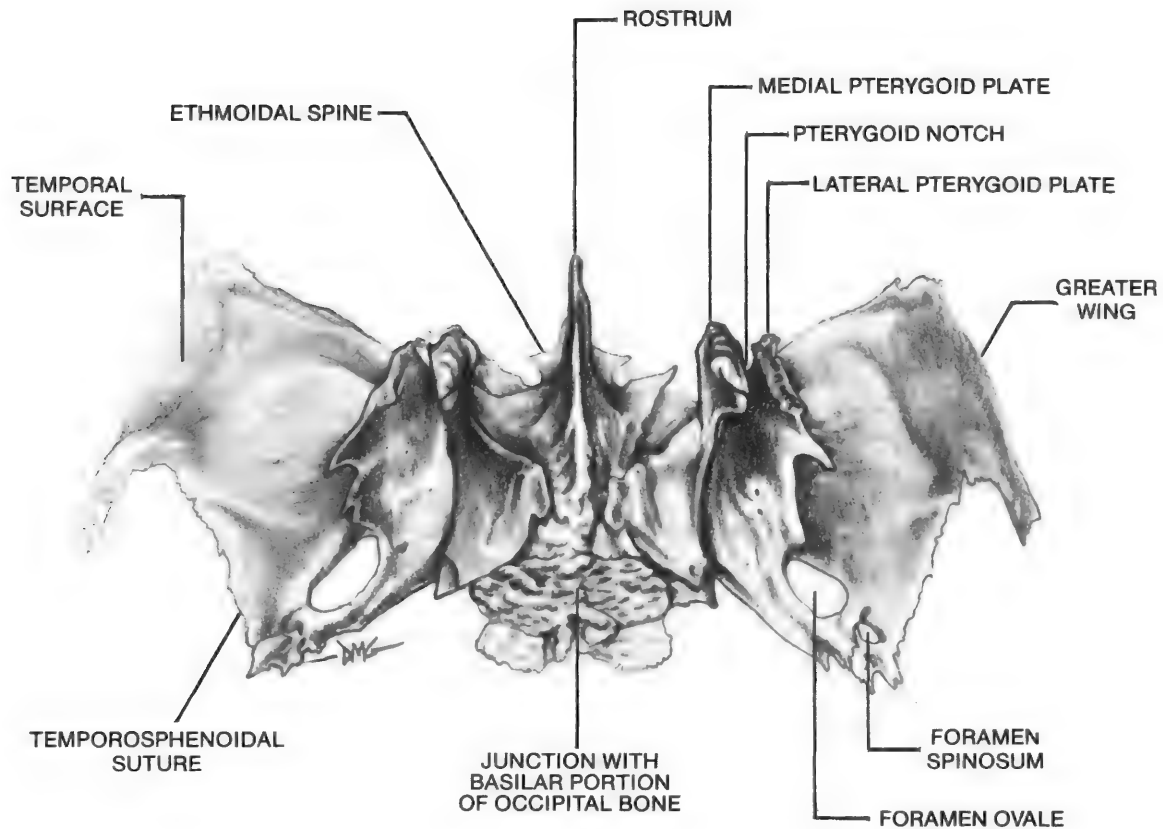
3—18. Sphenoid bone — superior view

Arising from the lateral aspect of the body are the *greater wings* of the sphenoid. These can be divided into the posterior part or floor, the temporal or lateral surface, and the orbital surface. At the anterior portion close to the base is the foramen rotundum, which contains the maxillary nerve. Posterior and lateral is the foramen ovale, which contains the mandibular nerve, the accessory meningeal artery, and sometimes the lesser petrosal nerve. Posterior and lateral to the foramen ovale is the foramen spinosum, which is much smaller and channels the middle meningeal vessels and recurrent branch from the mandibular nerve. The temporal surface is broad and flat, angling superiorly to continue articulating with the temporal squama, and then turning anteriorly at the junction of the parietal.

The orbital surface of the greater wing is

generally very thin and forms the posterolateral wall of the orbit. The superior aspect articulates with the orbital surface of the frontal bone, while the lateral margin articulates with the zygomatic bone. The medial margin forms the inferior lateral boundary of the superior orbital fissure. The inferior head of the lateral rectus muscle attaches to a tubercle in the middle portion of this margin, and the inferior border forms the posterolateral boundary of the inferior orbital fissure.

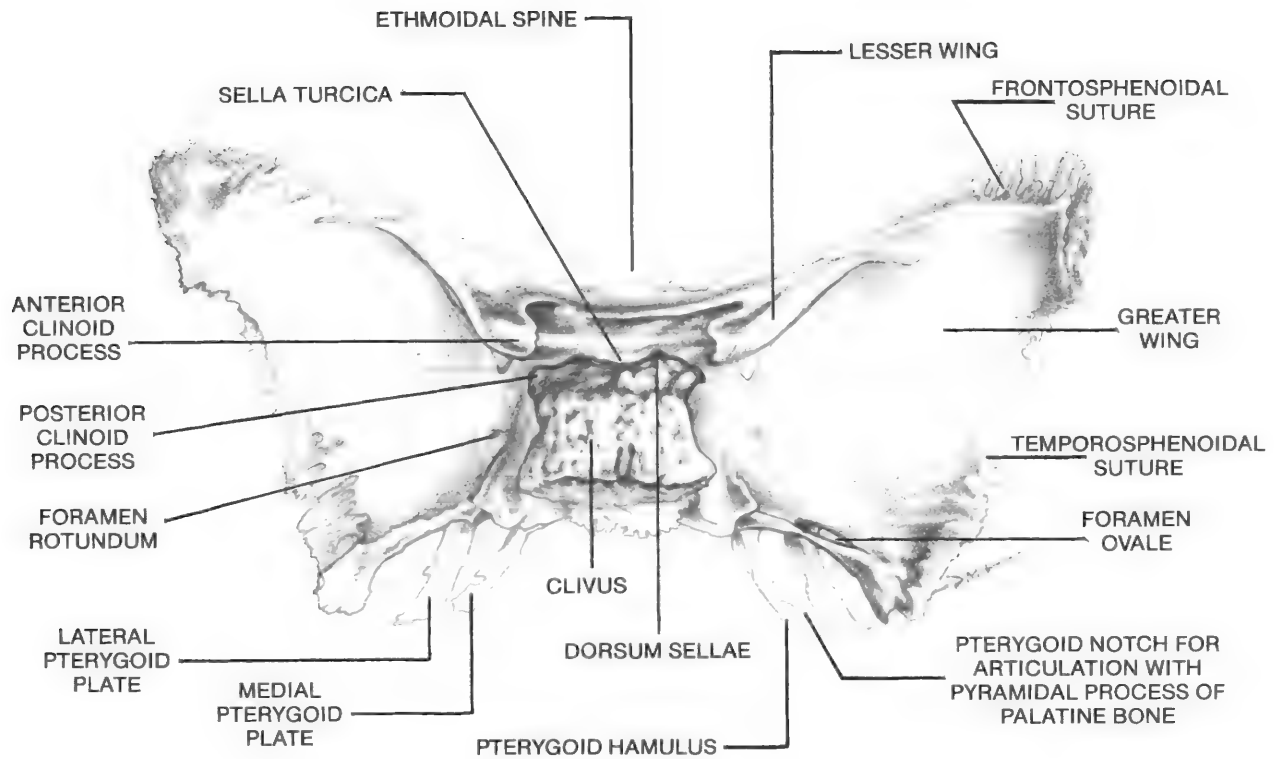
The *lesser wings* arise from the anterior superior aspect of the body and resemble two triangular projections ending in long, thin points. The anterior aspect of the lesser wings articulates with the frontal bone. The lesser wing is connected to the body of the sphenoid by two roots, whose division makes up the optic canal.



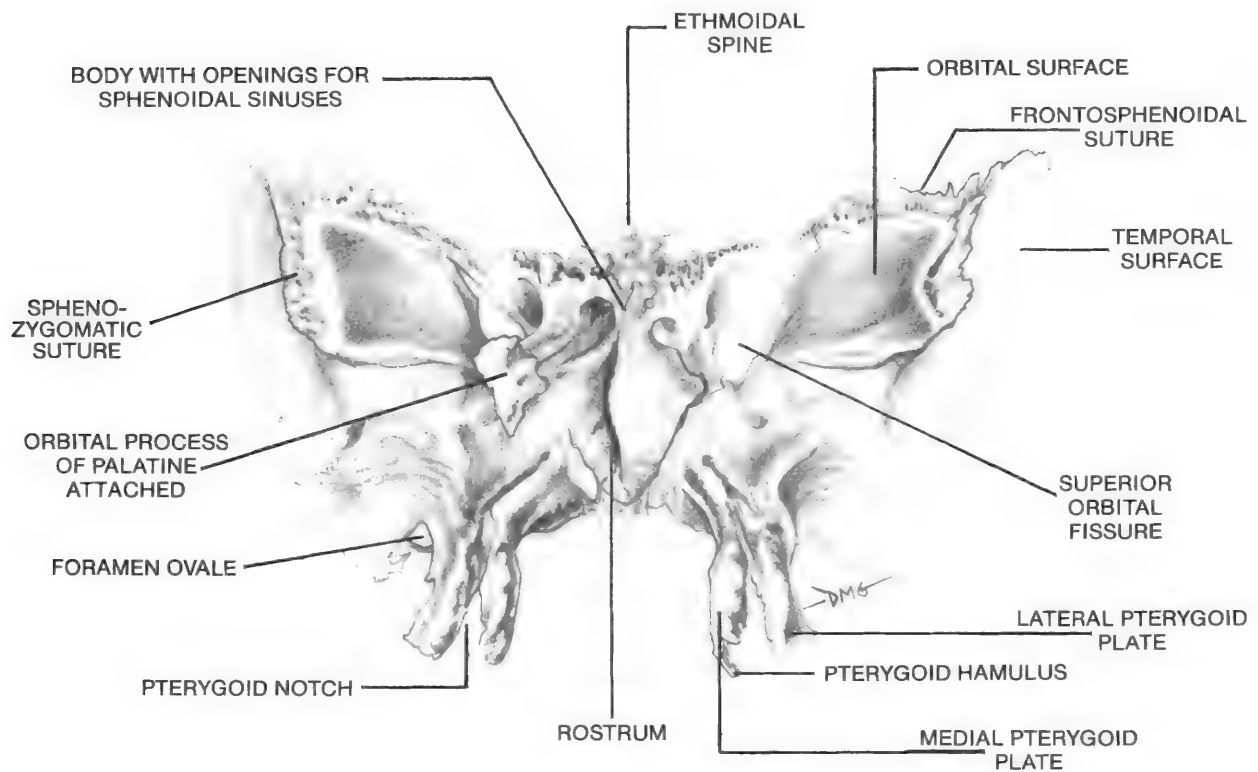
3—19. Sphenoid bone — inferior view

The *pterygoid processes* arise from the body and greater wing of the sphenoid, projecting inferiorly. The pterygoid processes divide inferiorly, forming the medial and lateral pterygoid plates, yielding the pterygoid fissure which articulates with the pyramidal process of the palatine bone. The lateral pterygoid plate is broad, thin, and everted, and makes up part of the infratemporal fossa. The inferior head of the external pterygoid muscle is attached to the inferior lateral projection, while to the medial lateral projection is attached the medial pterygoid muscle. The

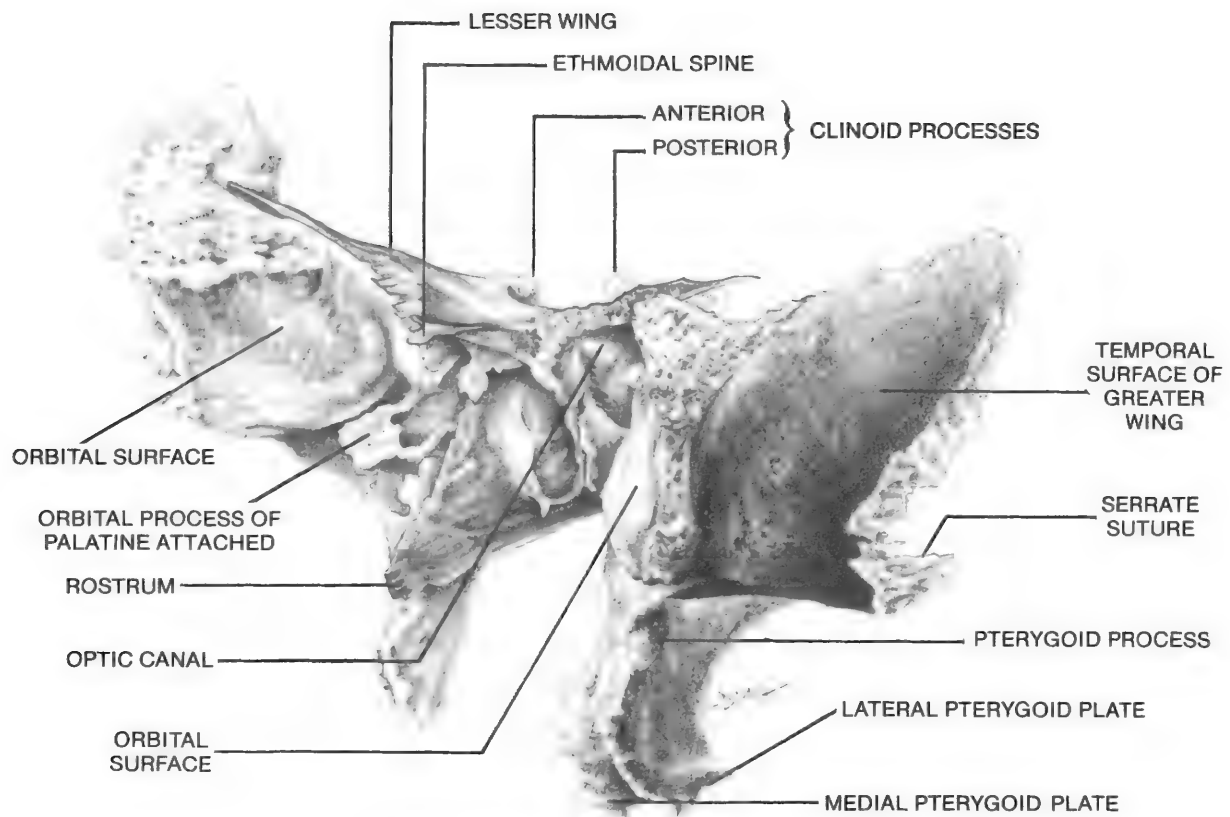
lateral projection of the pterygoid process is best reached for examination, challenge, and treatment when the physician's finger is inserted lateral to the teeth (on the buccal side) past the last molar and slightly medial. The medial pterygoid plate is narrow, longer, and has a hook-like process called the pterygoid hamulus at its inferior extremity. This part of the pterygoid process can best be reached for examination, challenge, and treatment by inserting the finger past the molar teeth on the medial (lingual) side to make contact with the inferior medial portion.



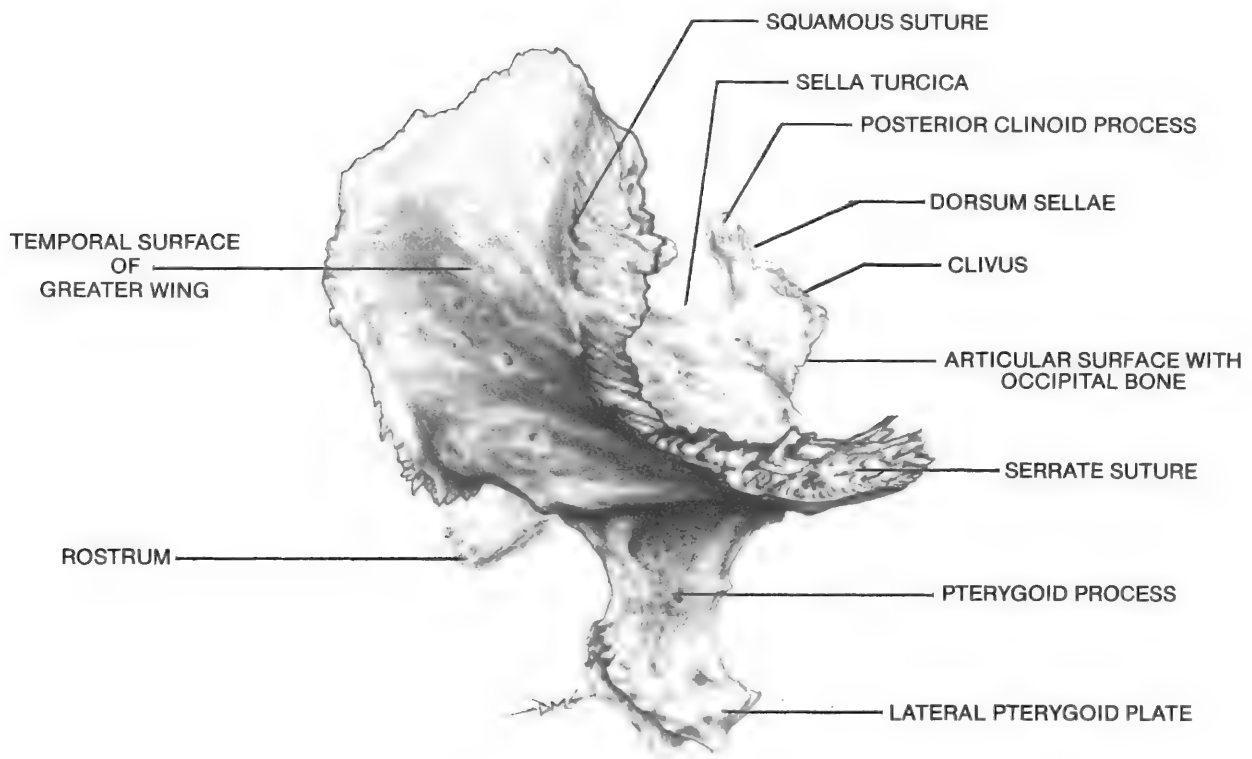
3—20. Sphenoid bone — posterior view



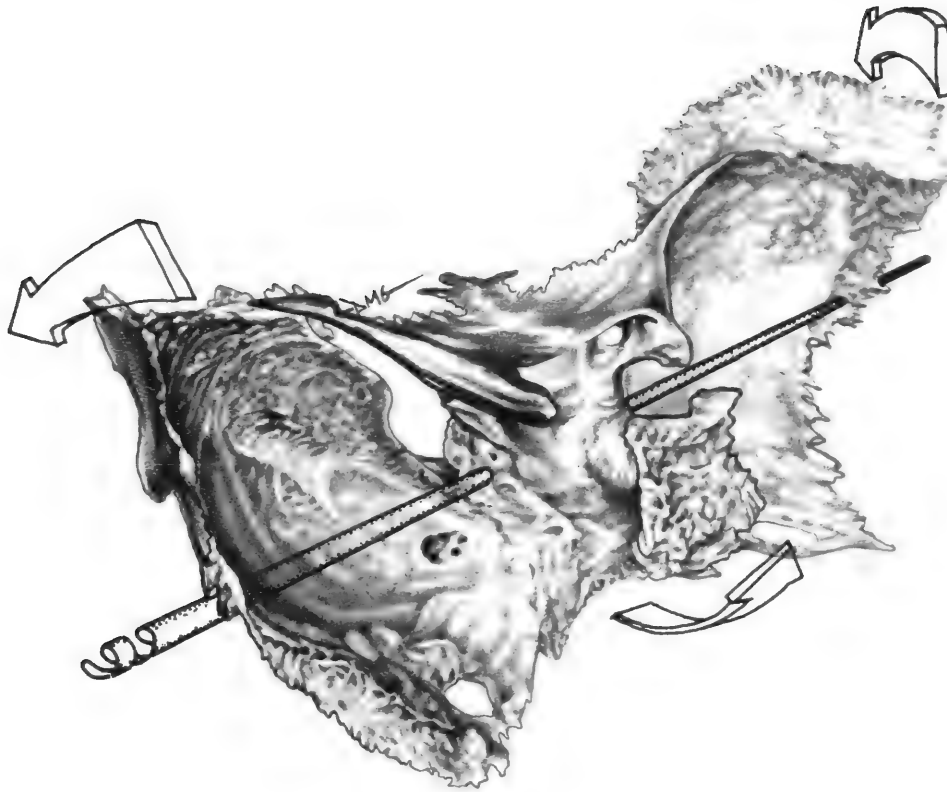
3—21. Sphenoid bone — anterior view



3—22. Sphenoid bone — anterolateral view



3—23. Sphenoid bone — lateral view



3—24. Superior posterolateral view of sphenoid with arrows showing motion on sphenobasilar flexion (inspiration). Line indicates general axis of rotation, through the sphenosquamous pivot.

Movement

The motion of the sphenoid is rotation in the transverse plane. The general axis of rotation is anterior and inferior to the sella turcica, in general alignment with the sphenosquamous pivot which will be discussed under the articulations of the bone.

Body: the posterior aspect of the body, articulating with the basilar portion of the occipital bone, moves superiorly and slightly posteriorly on sphenobasilar flexion (inspiration).

Greater wings: the superior portion of the greater wings moves anteriorly and laterally, while the inferior aspect moves posteriorly in sphenobasilar flexion.

Pterygoid processes: the pterygoid processes pri-

marily move posteriorly with slight lateral and inferior motion.

Rostrum: the anterior aspect of the rostrum moves inferiorly and slightly posteriorly.

Articulations

Sphenobasilar: the sphenobasilar junction is a synchondrosis until about the age of twenty-five, when it completes ossification to cancellous bone. The motion at this point is the key which correlates all other motions of the cranium. After ossification, the motion is that of flexion and extension of living bone, aided by the fact that this is cancellous bone.

Sphenopetrous is the body of the sphenoid articulating with the apex of the petrous portion of

the temporal bone. This synchondrosis, which usually does not ossify throughout life,²³ is between the petrosal process of the dorsum sellae and the apex of the petrous portion of the temporal bone. The motion on sphenobasilar flexion (inspiration) is a rocking, slightly twisting type as the petrosal process rises and the petrous apex moves anteriorly and laterally. The mobility of the articulation between the body of the sphenoid and the apex of the petrous portion of the temporal bone has a direct bearing on the amount of motion that will be available at the sphenobasilar junction.

Temporosphenoïdal suture: this suture must be divided for study, as the functions of the inferior section are different from those of the superior section. The division line is approximately in the middle and is called the sphenosquamous (SS) pivot.³⁶ A line from the right to left SS pivot is the general axis of rotation for the sphenoid bone.

The suture below the SS pivot is a serrate suture with significant interdigitation. During sphenobasilar flexion (inspiration), there is a rocking as the anterior inferior squama of the temporal bone moves slightly inferiorly and posteriorly, while the floor of the greater wing of the sphenoid bone moves slightly posteriorly and superiorly.

Above the sphenosquamous pivot, the suture is squamosal with an external bevel on the lateral aspect of the greater wing of the sphenoid. This shape gives a sliding capability to the junction between the lateral aspect of the sphenoid's greater wing and the temporal bone, which is necessary for the external rotation and slightly anterior and inferior movement of the temporal bone squama as it articulates with the greater wing of the sphenoid. The superior lateral wall of the greater wing of the sphenoid moves anteriorly, laterally, and slightly inferiorly with sphenobasilar flexion (inspiration).

The *sphenoparietal suture* is a widely beveled squamous suture with an internal bevel on the greater wing of the sphenoid bone. The superior aspect of the greater wing of the sphenoid moves

anteriorly, while the parietal bone externally rotates during sphenobasilar flexion. External rotation of the parietal bone means that the sphenoidal and temporal borders flare laterally.

Both the greater and lesser wings of the sphenoid have *frontosphenoïdal sutures*. The suture of the greater wing is L-shaped and has varying characteristics. The frontosphenoïdal suture associated with the lateral surface is primarily limbic, with the internal bevel on the sphenoid. This gives continued sliding action of the superior aspect of the wing of the sphenoid as it moves anteriorly on sphenobasilar flexion. The portion of the suture associated with the orbital surface is a serrate suture with significant interdigitation with the more stable frontal bone. The motion is of an expansile nature. The lesser wing of the sphenoid has a serrate suture from the body to the point of the lesser wing where it becomes significantly needlelike. This articulation with the orbital plate of the frontal gives an expansile-type motion similar to that of the orbital section of the greater wing. The end projections of the lesser wing are attached along the ridge of the orbital plate of the frontal bone.

The *sphenozygomatic suture* is a limbic suture, with the external bevel on the sphenoid. Motion is of expansion and contraction. As this area of the sphenoid moves anteriorly and slightly inferiorly, it carries the zygomatic bone with it.

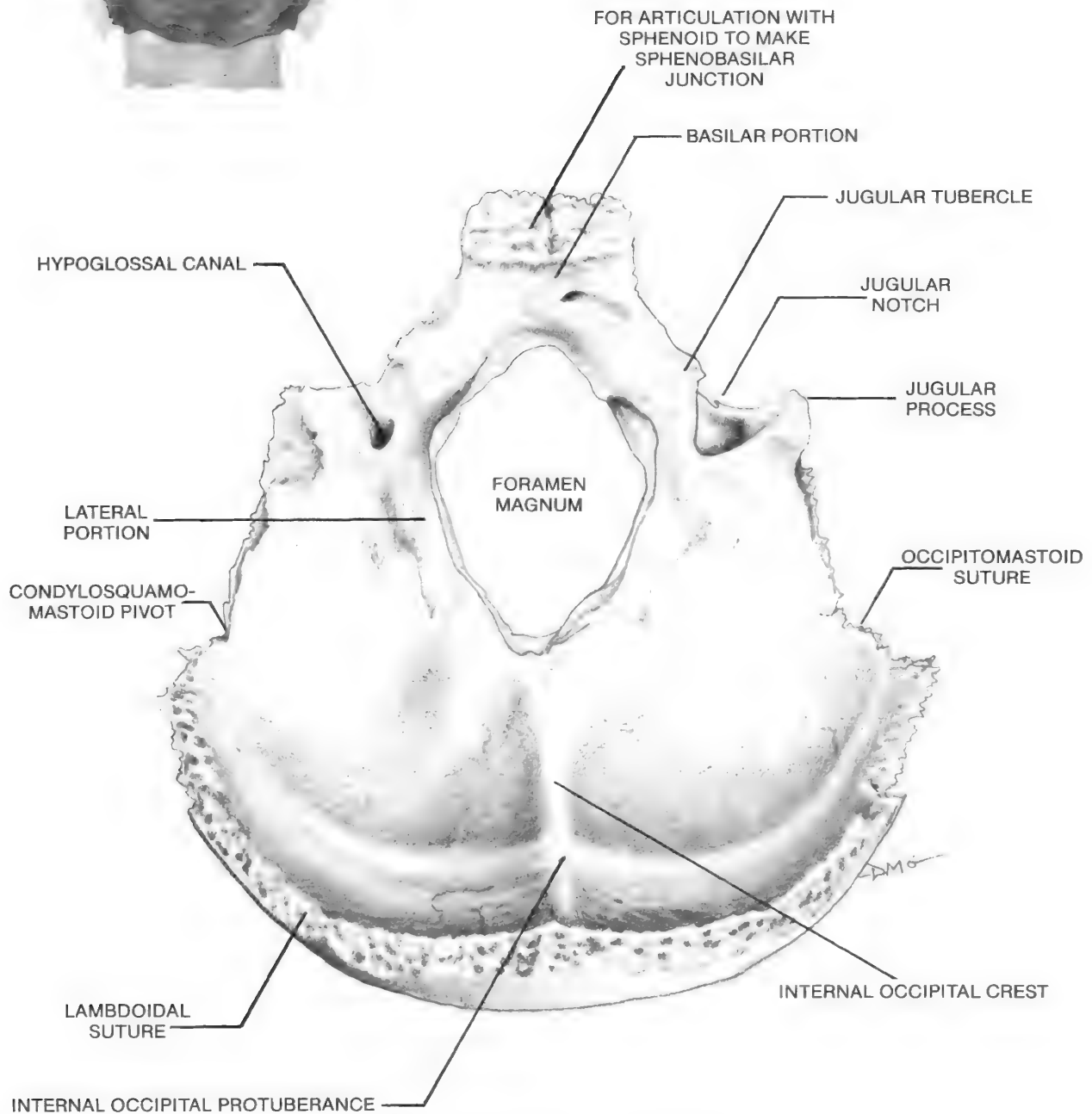
The palatine bones are joined to the pterygoid processes bilaterally at the infralateral surface of the body of the sphenoid anteriorly, and at the pterygoid processes inferiorly. These motions are discussed under the palatine bone.

The ethmoid is joined to the anterior body of the sphenoid and the sphenoidal crest in the anterior midline of the sphenoidal body. The sphenoidal concha articulates with the lamina orbitalis of the ethmoid.

The vomer articulates with the sphenoid by the rostrum and the vaginal processes of the medial pterygoid plates.

Occipital Bone

The occiput is the line of communication between the spinal column and the cranium. It is comprised of four parts which are divided by the foramen magnum. The large plate posterior to the foramen magnum is the squama; the thickened section anterior to the foramen is the basilar part; and on either side of the foramen are the lateral portions, sometimes called the interior parts.



3—25. Occipital bone — superior view

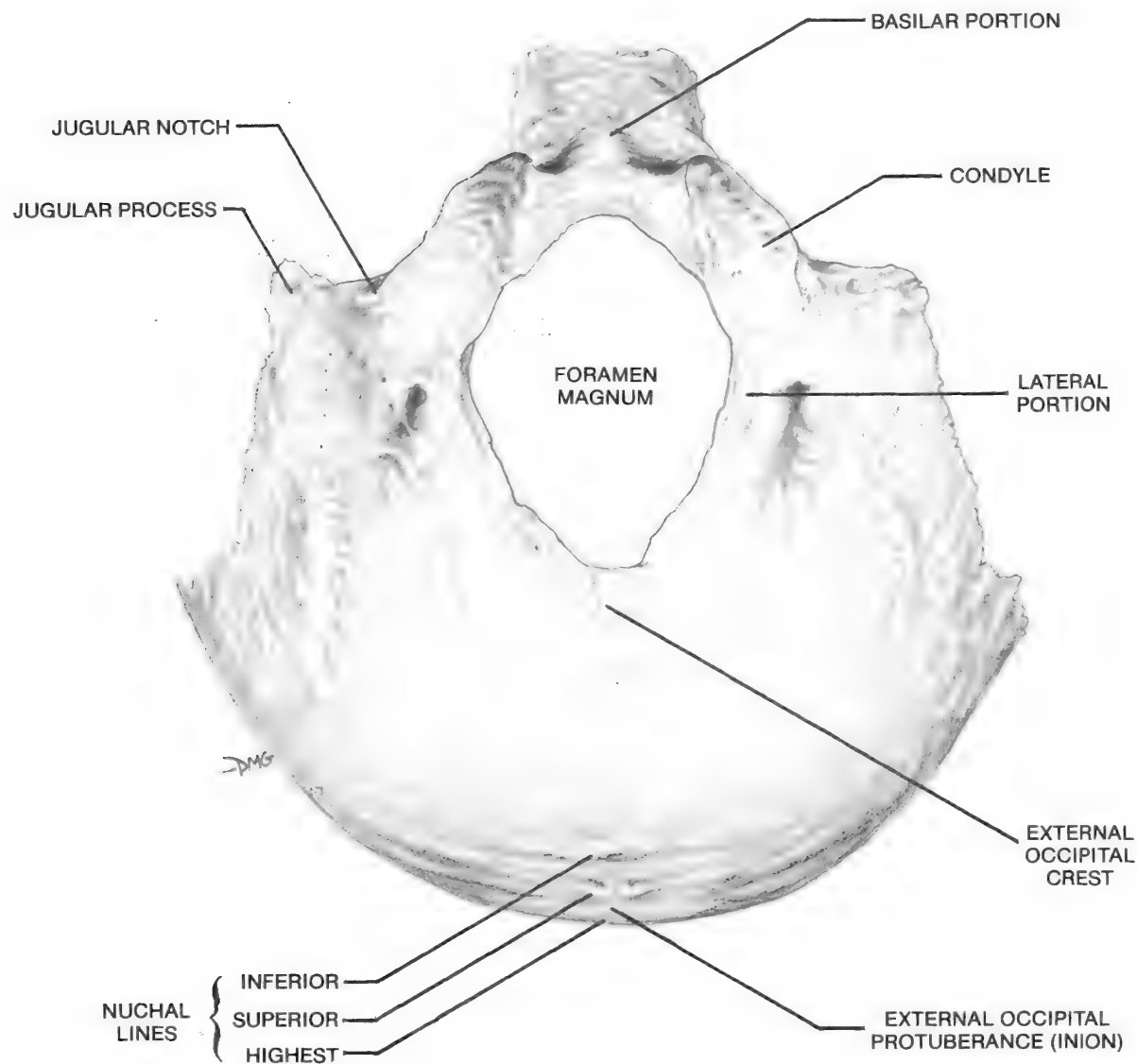
On the external surface of the *squama* are the highest, superior and inferior nuchal lines, along with the external occipital protuberance (inion). Numerous muscles important in head leveling and neurologic organization of the body are attached to the occipital squama.

The internal surface of the squama is divided by the cruciate eminence into four fossae. The occipital lobes of the cerebrum are located in the two superior fossae, while the hemispheres of the cerebellum are in the inferior fossae.

The *lateral parts* and *inferior surfaces* support the condyles for the articulation of the atlas vertebra. Extending laterally and somewhat superiorly is the

jugular process, which articulates with the temporal bone. The rectus capitis lateralis muscle originates from the jugular process. The jugular notch forms the posterior part of the jugular foramen. The hypoglossal canal, for exit of the hypoglossal nerve (cranial XII), is at the base of the condyle for the atlas.

The basilar portion, which is anterior to the foramen magnum, is thick and somewhat quadrilateral; it joins with the sphenoidal body. The articulation is cartilaginous and ossifies around the age of twenty-five. The rectus capitis anterior and longus capitis muscles originate from the external aspect of the basilar portion.



3—26. Occipital bone — inferior view

Cranial Osteology

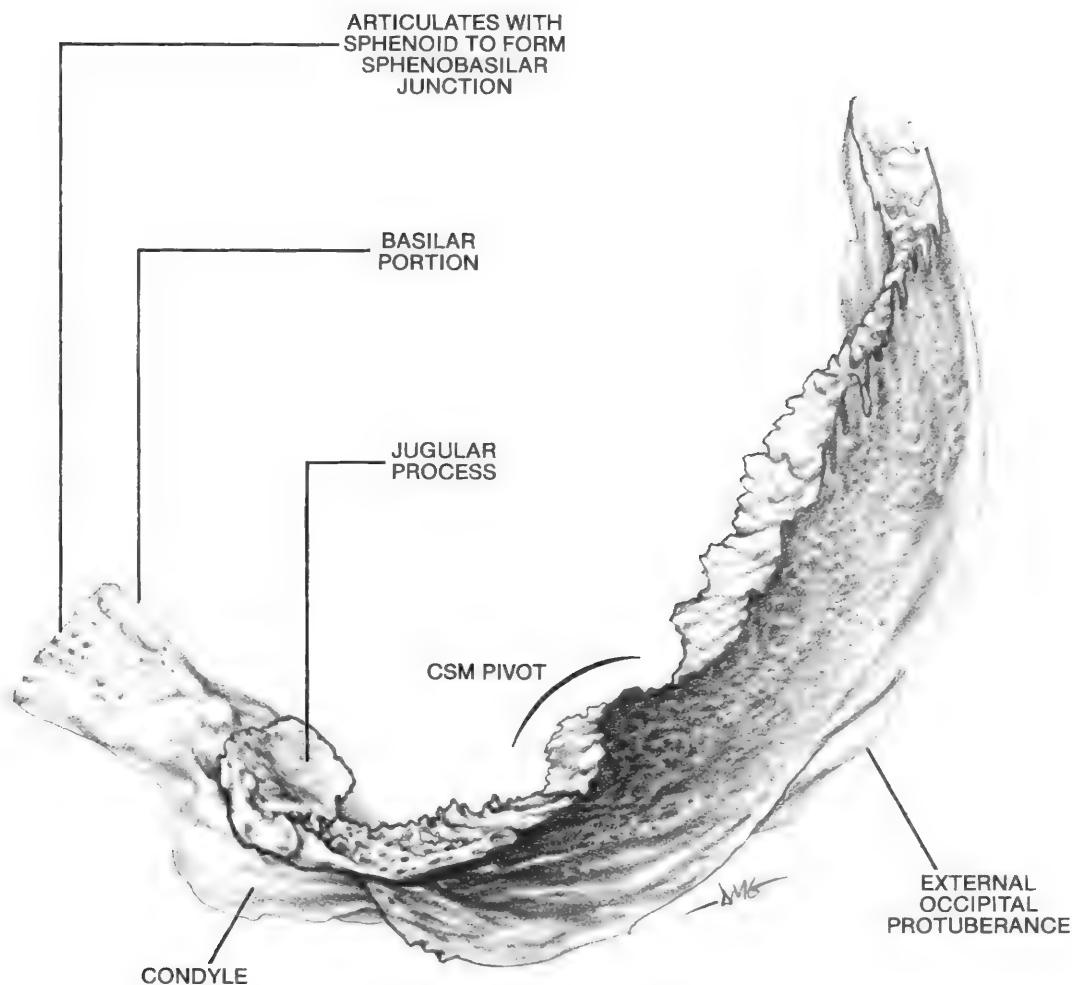
At birth there are four ossification centers for the occipital bone — one for the squama, two for the lateral portions, and one for the basilar portion. The occipital bone can be significantly distorted from direct trauma to the skull or muscular imbalances prior to complete ossification. If the faults or imbalances are not corrected, ossification of the occipital bone will be imbalanced; this is important in the consideration of cranial function. Improper ossification such as this is called an interosseous cranial fault. This creates structural distortion which will follow an individual throughout life, potentially creating further cranial as well as cervical stress and general neurologic disorganization throughout the body.

Movement

The motion of the occipital bone is that of flexion and extension in the sagittal plane. The basilar portion moves superiorly on sphenobasilar flexion (inspiration), while the superior aspect of the squama moves posteriorly and inferiorly.

Articulations

As previously mentioned, the basilar portion of the occipital bone articulates with the body of the sphenoid and is a synchondrosial suture, ossifying at approximately age twenty-five. From the point of ossification on, motion at the sphenobasilar junction is that of flexion and extension of living cancellous bone rather than that of a true articulation.



3—27. Occipital bone — lateral view

The lateral aspect of the basilar portion of the occipital bone articulates with the petrous portion of the temporal bone from the sphenobasilar junction to the notch for the jugular foramen. This is a relatively mobile synchondrosis which allows flexion and extension of the occipital and rotation of the temporal bone around its axis, located through the petrous portion. As the basilar aspect of the occipital bone rises, the apex of the petrous portion of the temporal bone moves anteriorly, laterally, and slightly superiorly, causing the posterior aspect of the apex end of the petrous portion to rise with the basilar portion of the occipital bone.

Posterior and lateral to the notch for the jugular foramen is the jugular process of the occipital bone, which articulates with the jugular surface of the temporal bone. The articulation is a synchondrosis which tends to ossify when there is lack of skull mobility.

From the jugular process to the asterion, the suture is known as the *occipitomastoid*. Approximately half-way along this sutural line, the suture turns sharply superiorly and its characteristic usually changes from that of an external bevel below to an internal bevel above the midpoint. The midpoint is known as the *condylosquamomastoid pivot* (CSM). The suture's characteristic is generally of less serrated interdigitation between the occipital and the temporal below the CSM pivot, and greater interdigitation above. The motion between the temporal and the occipital bones above the CSM pivot is in

opposite directions, with the interdigitations of the suture acting like teeth in a gear train. This is discussed later with the interrelation of skull bone motion.

The *lambdoidal suture* is composed of the occipital and parietal bones, between the asterion and the lambda. The lower lateral portion of the suture has an internal bevel on the parietal bone which accommodates the lateral flaring of the parietal on sphenobasilar flexion (inspiration). At a location that varies from skull to skull, the suture changes from a bevel to a greater serrated interdigitation. In some cases, the entire length of the suture is serrated, or even tends toward a dentate nature. The motion from here to the lambda is that of expansion and compression.

There is a great variance between skulls in the nature of the suture from the jugular process to the lambda. This normally appears to be a very mobile area. It is a location where there are many wormian bones with great sutural variation. This is also an area where there is controversy regarding the movement between the different concepts in cranial evaluation. The many differing opinions regarding this area may be because the structure varies from skull to skull. It appears there is a change in ossification, depending upon what is needed for maximum motion at the time of fontanelle closure and sutural development.

Temporal Bone

The temporal bone has three basic sections: the squama, the petrous, and the tympanic. Sometimes the mastoid portion and the styloid process are also considered as sections of the temporal bone.

The *squama* is the flat, broad portion making up the anterior and superior parts of the bone. Arising from the lower, somewhat anterior portion is the zygomatic process, which articulates with the zygomatic bone. The mandibular fossa is at the root end of the zygomatic process on the inferior aspect. This receives the condyle of the mandible to form the temporomandibular articulation. Immediately anterior to the mandibular fossa is the articular tubercle, which is a rounded eminence forming the anterior boundary of the mandibular fossa. At the posterior aspect of the squama — the junction with the mastoid portion — is the parietal notch, which articulates with the mastoid angle of the parietal bone.



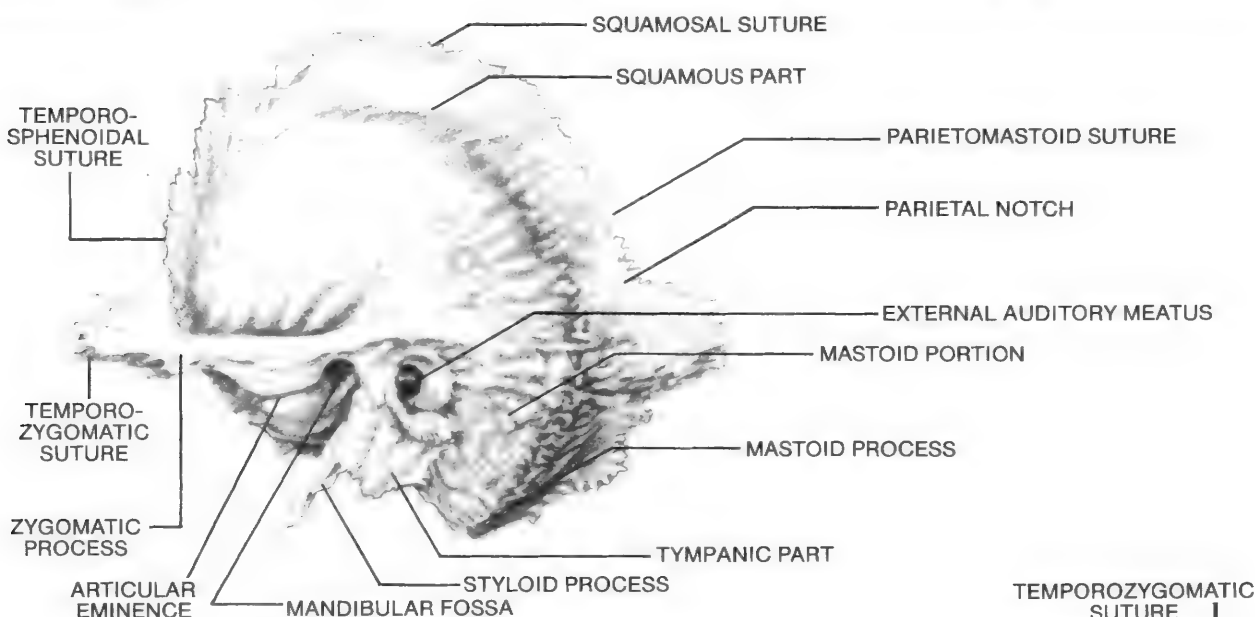
The *mastoid portion* forms the posterior portion of the temporal bone. The inferior projection from the mastoid portion is the *mastoid process*, which is

Cranial Osteology

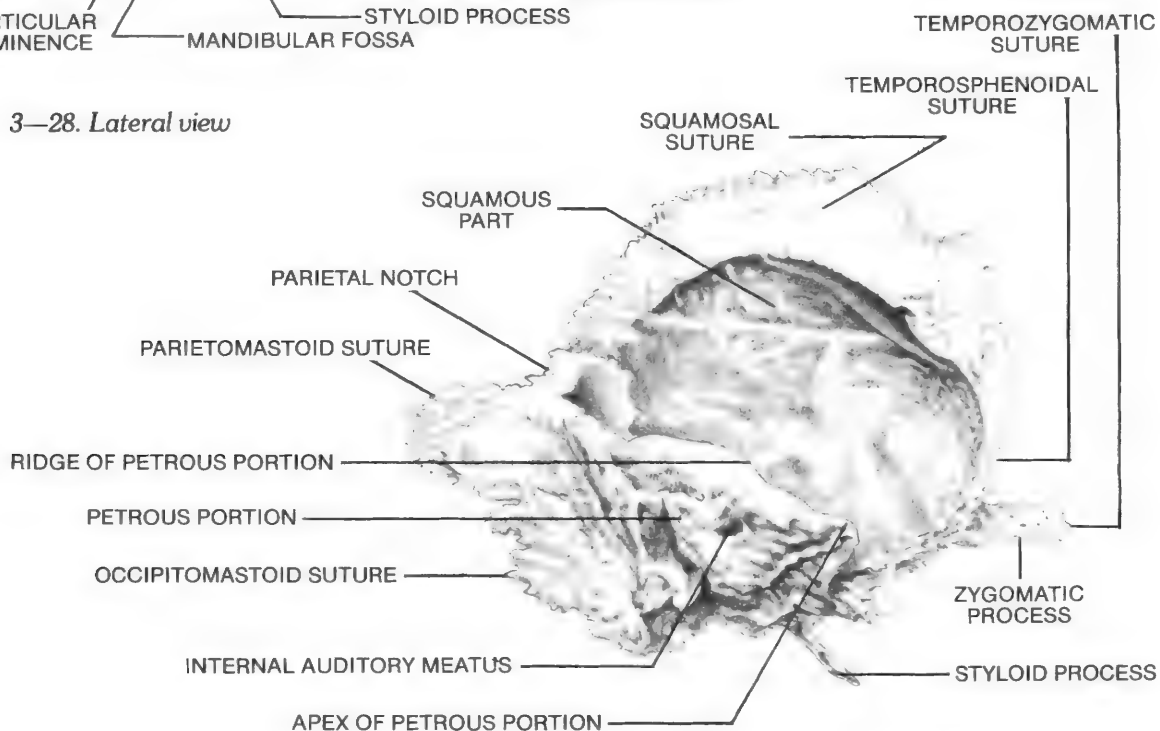
the attachment for the sternocleidomastoid, splenius capitis, and longissimus capitis muscles. In cranial terminology, one must be specific in stating the area of the mastoid being discussed. The motion of the mastoid portion differs from the motion of the mastoid process since the general axis of rotation of the temporal bone lies between these two areas. Much confusion in understanding cranial motion has developed because of not clearly stating the specific area being referenced. Medial to the mastoid process is the mastoid notch, sometimes called the "digastric notch," for the origin of the posterior belly of the digastric muscle.

The *petrous portion* extends medially and anteriorly from the squama. In the transverse plane it is almost level, dropping slightly inferiorly as it projects anteriorly. The base of the petrous portion originates from the squamous and mastoid portions of the temporal bone, giving rise to the apex which projects between the greater wing of the sphenoid and the basilar process of the occipital bone.

The *jugular foramen* is located between the petrous portion of the temporal bone and the jugular process of the occipital bone. The petrous portion forms the posterolateral boundary for the *foramen lacerum*, while anteriorly it is bounded by the body of



3—28. Lateral view



3—29. Medial view

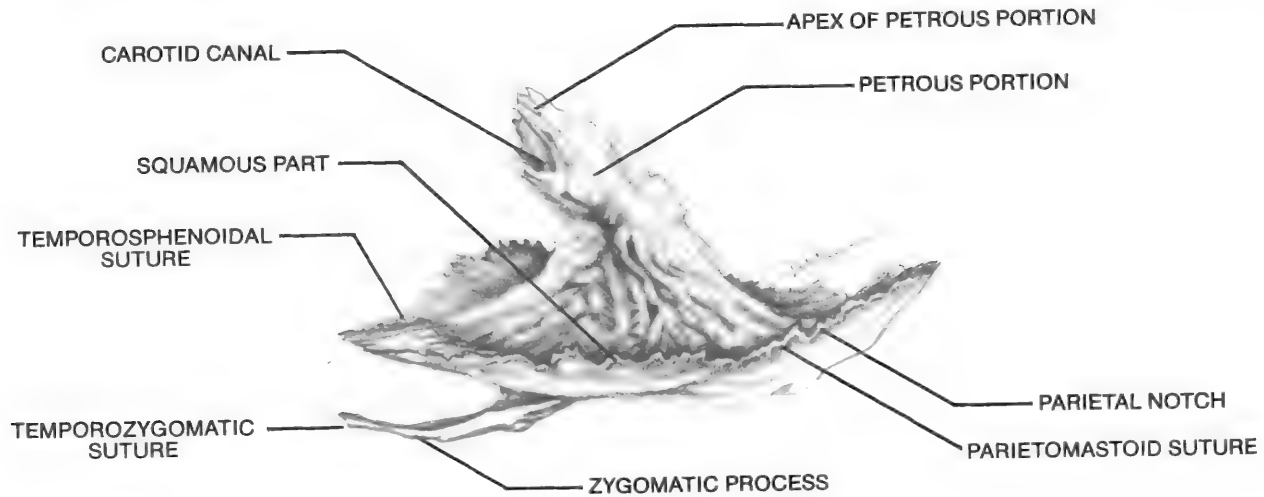
the sphenoid and posterior border of its greater wing. In young individuals where there is still significant mobility of the sphenobasilar junction, the basilar portion of the occipital bone is also involved with the foramen lacerum. Thus these important foramina are made up of two or three bones, allowing potential for disturbed mobility that may cause interference with the structures contained therein. The foramen lacerum makes up the anterior or internal orifice of the carotid canal.

The *internal acoustic meatus* is on the posterior surface of the petrous portion. Through this opening the facial and acoustic nerves, the nervus inter-

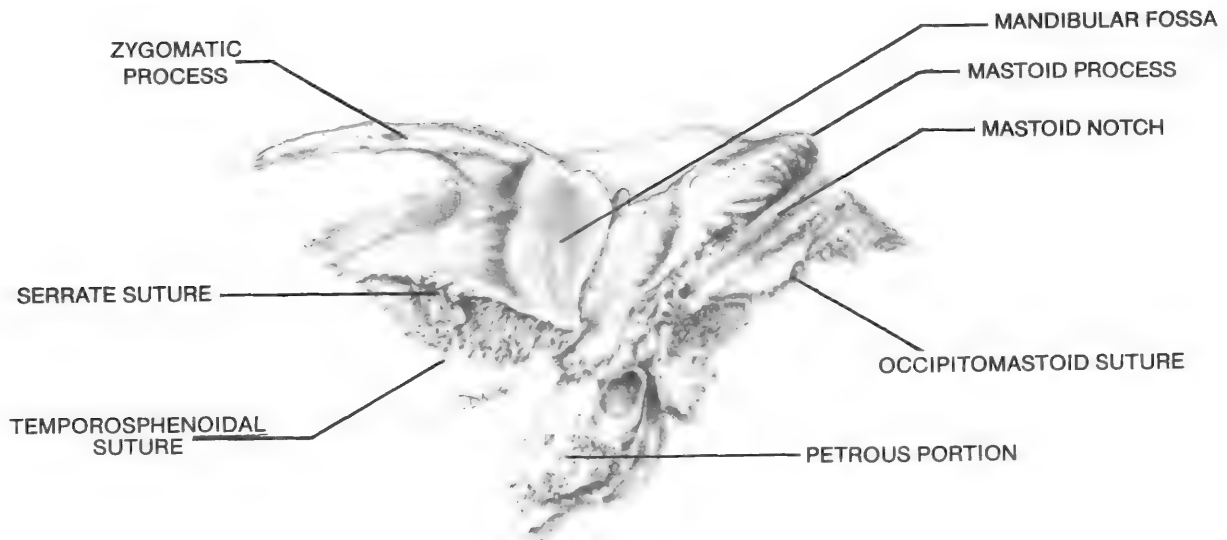
medius, and the internal auditory branch of the basilar artery are channeled into the internal acoustic canal.

The *tympanic portion* is a curved plate of bone lying inferior to the squama and anterior to the mastoid process. It makes up the posterior border of the mandibular fossa. The lateral border is the attachment point for the cartilaginous part of the external acoustic meatus. From the inferior portion arises the *styloid process*, which provides attachment for the styloglossus, stylohyoid, and stylopharyngeal muscles.

The temporal bone is extremely important in total cranial movement. It is an important link



3—30. Superior view



3—31. Inferior view

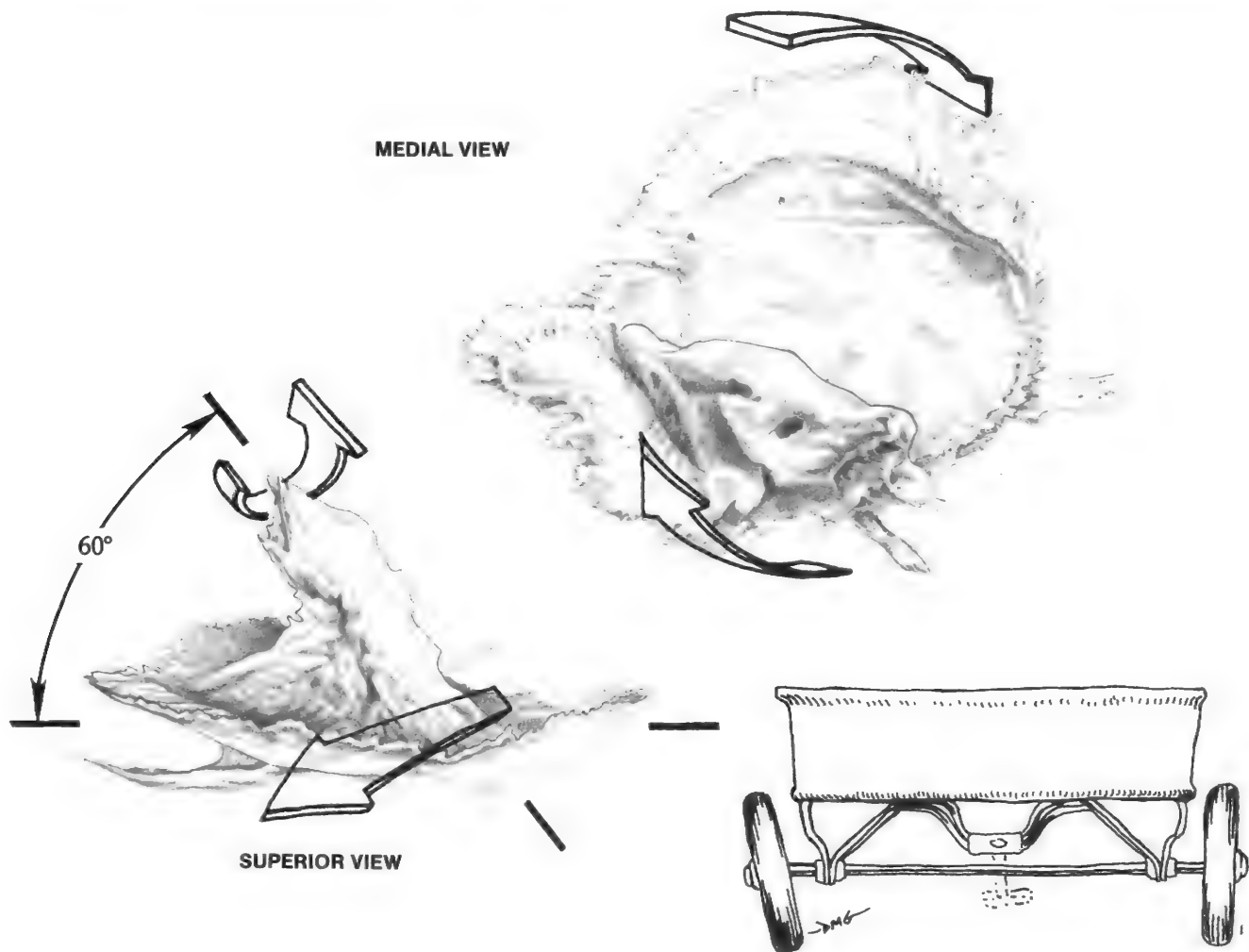
Cranial Osteology

between the sphenoid and occipital bones in relation to sphenobasilar flexion and extension. Its role is closely integrated with many of the symptomatic pictures which develop from cranial faults. Many stresses are placed upon it, and it is a key bone in cranial evaluation and correction.

Movement

The general axis of rotation of the temporal bone is through the petrous portion. It must be remembered whenever an axis of rotation is mentioned in cranial function that there are no true axes of rotation, just general areas. Cranial function depends upon the flexibility of living bone, as well as the minute movement between sutures. The motion, generally around the axis of the petrous portion of

the bone, combines rotation about a sagittal and a coronal axis; Sutherland has referred to this as a wobbling wheel effect.³⁶ Rotation about the coronal axis on sphenobasilar flexion (inspiration) is that of the superior portion of the squama moving anteriorly and the tip of the mastoid process moving posteriorly, while rotation about the sagittal axis is that of the superior squamous portion moving laterally and the tip of the mastoid process moving medially. There has been some confusion regarding this motion because of lack of communication or understanding of terminology. When the direction of mastoid rotation is discussed, it should be stated specifically whether reference is being made to the mastoid portion or the mastoid process and its tip. The mastoid portion of the temporal bone moves anteriorly



3—32. The axis of movement for the temporal bone on sphenobasilar flexion (inspiration) is indicated by the line through the petrous portion. The apex of the petrous portion moves anteriorly and laterally (external rotation), causing the structures which are joined to the axis of rotation at an angle to move as if a "wobbling wheel." The superior squama moves anteriorly and laterally, while the tip of the mastoid process moves posteriorly and medially.

and laterally with sphenobasilar flexion, while the tip of the mastoid process goes posteriorly and medially. Another portion of the temporal bone, in reference to motion, is the superior ridge of the petrous portion, which goes anteriorly and laterally on sphenobasilar flexion. This motion is sometimes referred to as "external rotation."

The sutures around the temporal bone are of the shape and type required for motion of the bone as described. Because the temporal bone is so important in examination, challenge, and correction in applied kinesiology, it is very important to thoroughly understand its movements. It is also very important to understand the temporal bone because of the monumental role it plays in creating various symptoms as a result of cranial primary respiratory dysfunction.

Articulations

A progressive trip around the articulations of the temporal with its adjacent bones is of value. During this excursion, the shape of the suture and its direct motion with the adjacent bone will be discussed, beginning with the joining of the occipital and temporal bones at the jugular surface of the temporal bone and the jugular process of the occipital bone.

The jugular process surface of the temporal bone is basically a flat, quadrilateral one covered with cartilage in the intact body. It is a synchondrosis which probably ossifies in direct relation with the mobility of the total skull. In some individuals with excellent skull mobility, it may well remain a synchondrosis throughout life. Whether or not it

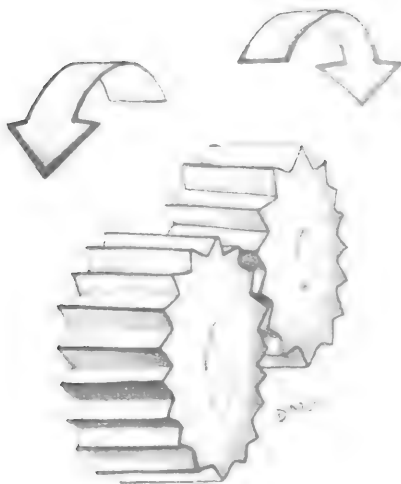
ossifies, it is a pivot point for movement between the temporal and occipital bones. Magoun²³ relates that Sutherland referred to it as a "... rock shaft and pivot for the rotation and undulation of the petrous."

From the jugular surface, the articulation progresses posteriorly and slightly superiorly and is called the *occipitomastoid suture*. The sutural surface is slightly serrated, with some interdigitation at this area. The suture turns more superiorly at the *condylosquamomastoid (CSM) pivot*. Often there is an external bevel inferior and an internal bevel superior to the condylosquamomastoid pivot. The suture from the CSM pivot to the asterion has more significant projections acting as teeth on a gear as they interdigitate with the occipital bone. The motion in this location is in opposite directions, acting as the periphery of two gears functioning together. On sphenobasilar flexion (inspiration), this portion of the occipitomastoid suture moves superiorly and separates slightly, while the portion of the suture below the CSM pivot tightens.

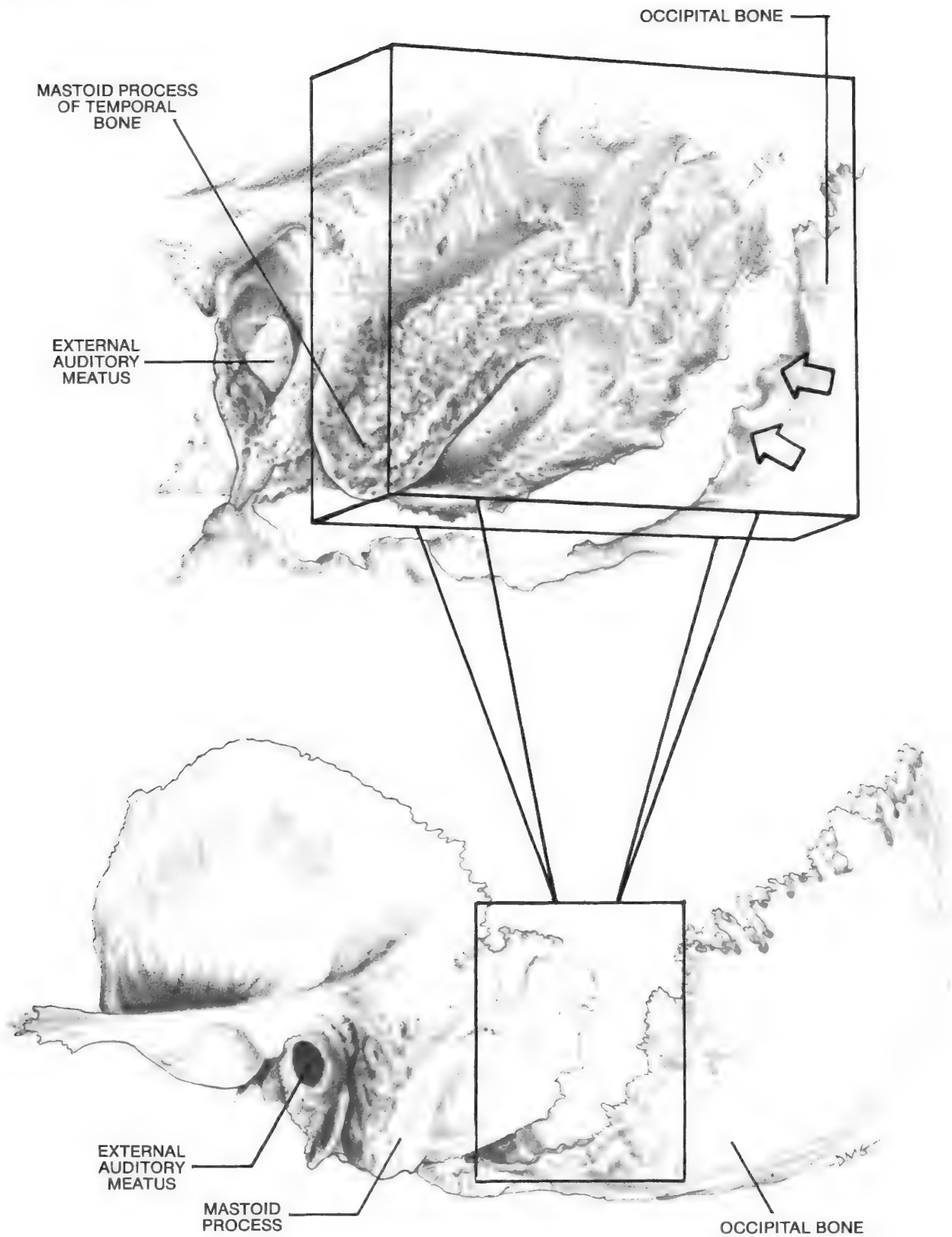
From the asterion, the articulation projects anteriorly for approximately 2 cm, forming the *parietomastoid suture*. This is a serrate suture giving significant interdigitation between the parietal and temporal bones at the parietal notch. It is a pivot point for the rotational factors of these bones.

Wormian bones are often found in the area of the asterion, parietal notch, and the superior occipitomastoid suture. Since it is extremely important for motion to be present in this area and there is potential for jamming, it seems reasonable that these bones may sometimes be formed during ossification as an effort of the body to maintain motion. This would seem to be a realistic adaptation if cranial stresses were present during growth.

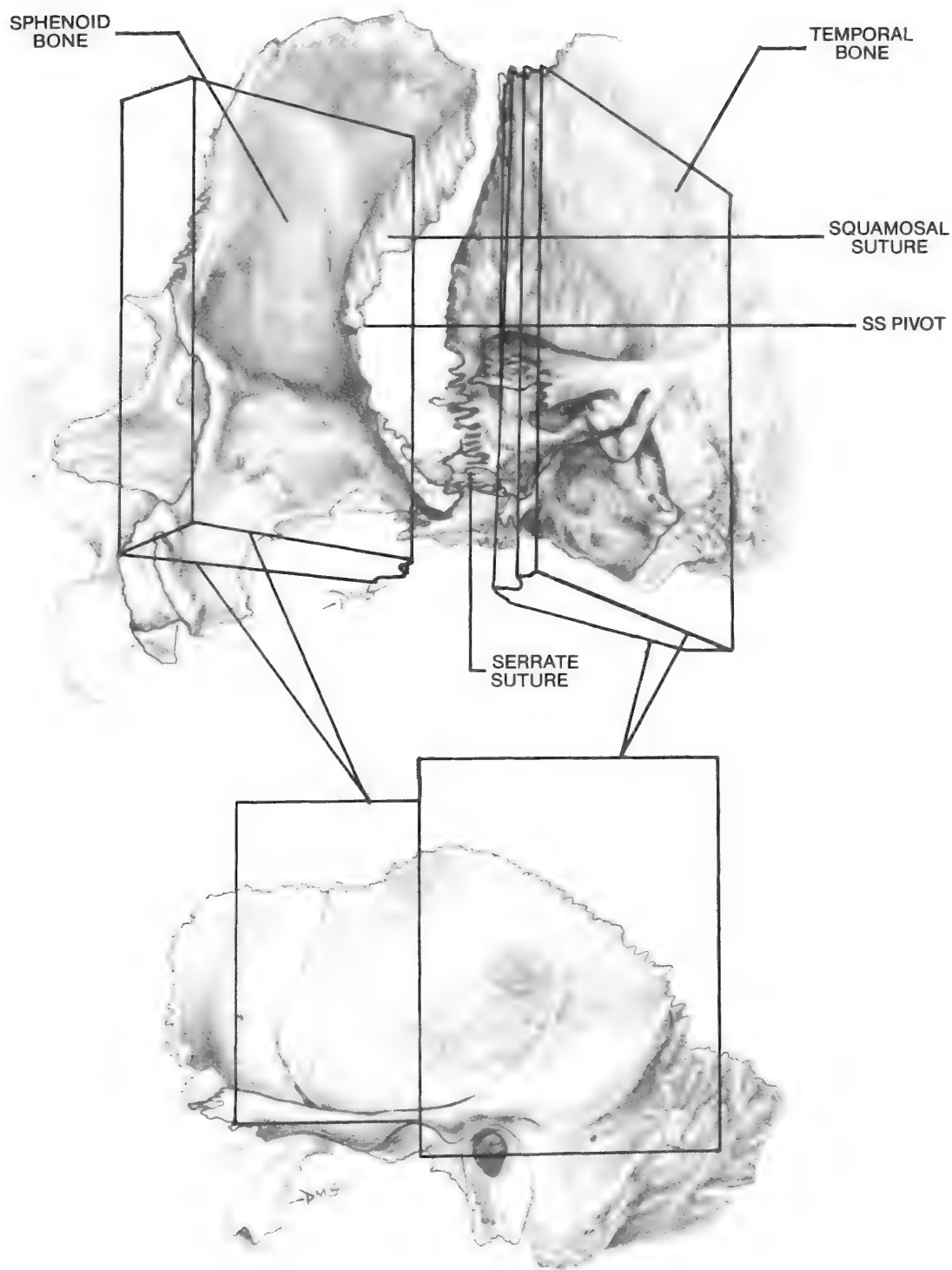
The suture dramatically changes to a squamous suture at the anterior portion of the parietal notch; it is then called the *squamosal suture*. It is identified externally by the inferior temporal line, which marks the posterior border of the temporalis muscle. The squamous suture extends around the temporal bone to the greater wing of the sphenoid. As the temporal joins with the sphenoid, the suture becomes the *temporosphenoidal suture* and maintains its broad squamous appearance with no more than a line of demarcation at the change of bones. At approximately the level of the superior border of the zygomatic arch, the suture dramatically changes from squamous to serrated, and from internal to external bevel. The change in the suture is called the *sphenosquamous (SS) pivot*. The unique flat, broad, internally bevelled squamous suture of the temporal bone above this point bestows a gliding action on the temporal with the parietal and sphenoid bones. This mechanical feature allows the temporal bone to



3—33. Gear-like mechanism of the occipitomastoid suture above the condylosquamomastoid (CSM) pivot.



3—34. There is deep, solid interdigitation at the occipitomastoid suture indicated with arrows in top illustration.



3—35. Motion on sphenobasilar flexion (inspiration) is sliding above the SS pivot and expansion and contraction below. Enlarged illustration shows the bones separated and turned to reveal the nature of the different areas of the sutures.

Cranial Osteology

rotate in both the sagittal and transverse planes.

As the suture progresses inferiorly from the SS point, it changes to serrated interdigitation between the temporal and sphenoid bones. This changing characteristic of the temporosphenoidal suture is very important in understanding the motion which takes place on sphenobasilar flexion and extension and the challenges which are used in applied kinesiology to evaluate this entire structural complex. The general axis of rotation of the sphenoid is just anterior to the sella turcica, in alignment with the sphenosquamous pivot of the temporosphenoidal suture. The location of the general axis of rotation may vary somewhat and is regulated by the location of the SS pivot. During sphenobasilar flexion there is a slight separation of the temporosphenoidal suture, which is above the horizontal axis of rotation of the sphenoid, and a rocking motion of the more interdigitated suture below the axis of rotation.

During sphenobasilar extension (expiration), there is a sliding of the temporosphenoidal suture with the greater wing of the sphenoid, and a slight rocking of the interdigitated suture below the axis of rotation. Remember the mechanics of the temporosphenoidal suture when evaluating the motions which are imparted by applied kinesiology challenge, and by therapeutic pressures of inspiration and expiration assist applied to the tip of the mastoid process.

The petrous portion of the temporal articulates with the floor of the greater wing of the sphenoid anteriorly, and with the basilar portion of the occipital bone posteriorly. The apex of the petrous portion articulates with the body of the sphenoid. These articulations are synchondroses and may well be cartilaginous throughout life. They are extremely important because they enable the temporal bone to rotate around its general axis, which is through the petrous portion. This motion is also important because of the relationship of the petrous portion of the temporal bone with the jugular and lacerum foramina.

Study of the temporal bone shows that it is highly movable in relation to the cranial motion concept. It is also extremely important because of its link with the sphenoid and occipital bones. An imbalance of the temporal bone influences the sphenoid, the key

bone in all cranial dysfunction because of its many articulating surfaces with other bones. The temporal also influences the occipital bone, the connecting articulation with the spinal column. Because of the temporal's intricate relation with these key bones, stresses and pressures upon it are exceptionally important in either creating or correcting cranial faults.

Stresses which can create cranial faults related to the temporal bone are muscular imbalances which can result from trauma or functional disturbances. The muscles directly associated with the temporal are of postural relation, temporomandibular joint function, and hyoid muscles. Postural muscles are the sternocleidomastoid, splenius capitis, and longissimus capitis; muscles of mastication are the masseter and temporalis. The muscles of the hyoid are the stylohyoid and posterior belly of the digastric. The temporal bone is also involved with the styloglossus, auricularis group, temporoparietalis, and occipitalis muscles.

The temporal bone may sometimes receive great stress from improper function of the temporomandibular articulation. If an individual's muscles of mastication are off-balance, mastication will put mechanical stress of an imbalanced nature into the bilateral temporal bones. On the other hand, the muscles may be balanced but dental procedures or a change in dentition may create a poor occlusion, which causes imbalanced forces to be imparted to the temporal bone every time the patient bites. It must be remembered that there is tremendous force generated in chewing. The muscles of mastication have sufficient strength to support the body's weight from the grip of the teeth.

The temporal bones continue to play an important role in body dysfunction by forming half of the jugular foramen, through which must pass the inferior petrosal and transverse sinuses, the internal jugular vein, and the glossopharyngeal, vagus, and accessory nerves. The foramen lacerum has a similar status, leading to the carotid canal for the internal carotid artery.

The temporal bone is involved with the organs of hearing and equilibrium. These can be disturbed from circulatory, neurologic, or positional problems caused by cranial faults.

Frontal Bone

The frontal bone consists of two sections, the vertical portion, which corresponds with the forehead, and the orbital or horizontal portion, which enters into the formation of the roofs of the orbital and nasal cavities.

The external surface of the squama is convex; it is formed by two ossification centers, one for each half. Before ossification is complete, there is a division of the two halves of the bone called the metopic suture. It generally ossifies, leaving only a remnant of the suture at the inferior border of the frontal bone. Bilaterally there is an eminence — the frontal eminence — about 3 cm superior to the supraorbital margin. The prominent margin making up the superior orbit is the supraorbital margin, above which is the superciliary arch. Between the superciliary arches there is a smooth elevation called the *glabella*. The supraorbital margin has a notch or foramen through which pass the supraorbital vessels and nerve. The supraorbital margin extends into the zygomatic process, which articulates with the zygomatic bone. Arising from the zygomatic process is the temporal line. Below the glabella is the nasal notch, articulating with the nasal and lacrimal bones, frontal processes of the maxilla, and the nasal spine, which helps form the nasal cavity and the septum of the nose. It articulates with the nasal bones and the perpendicular plate of the ethmoid.

The internal surface has in its midline a vertical groove called the sagittal sulcus, which ends at the frontal crest. The groove contains the sagittal sinus, and the falx cerebri is attached to its margins.

The orbital or horizontal part is primarily thin plates of bone which form the superior aspect of the orbit. They are separated from each other by the ethmoidal notch, into which fits the cribriform plate of the ethmoid. Openings in the frontal bone communicate with openings in the ethmoid to complete ethmoidal air cells.

Movement

Normal cranial motion is dependent upon frontal bone motion. The frontal bone, however, does not have the same range of motion as other cranial structures. It depends upon the flexibility of living bone to move with other cranial structures.

The motion of the frontal bone is referred to as internal and external rotation. Unfortunately, the portion of the bone being referred to has varied in different systems of evaluation and treatment. In cranial osteopathy and DeJarnette's sacro occipital technique, the term "external rotation" refers to the lateral aspect of the squamous portion of the frontal



bone externally rotating with the temporal bone during sphenobasilar flexion. In applied kinesiology, external rotation refers to the metopic suture area, which in turn would be internal rotation of the lateral squamous portion of the frontal bone. On sphenobasilar extension, the reverse is true. In applied kinesiology, sphenobasilar extension is equivalent to external rotation of the metopic suture area, and internal rotation of the squamous portion of the frontal bone. In this text, internal and external rotation of the frontal bone will be equated with the particular portion of the frontal bone being discussed.

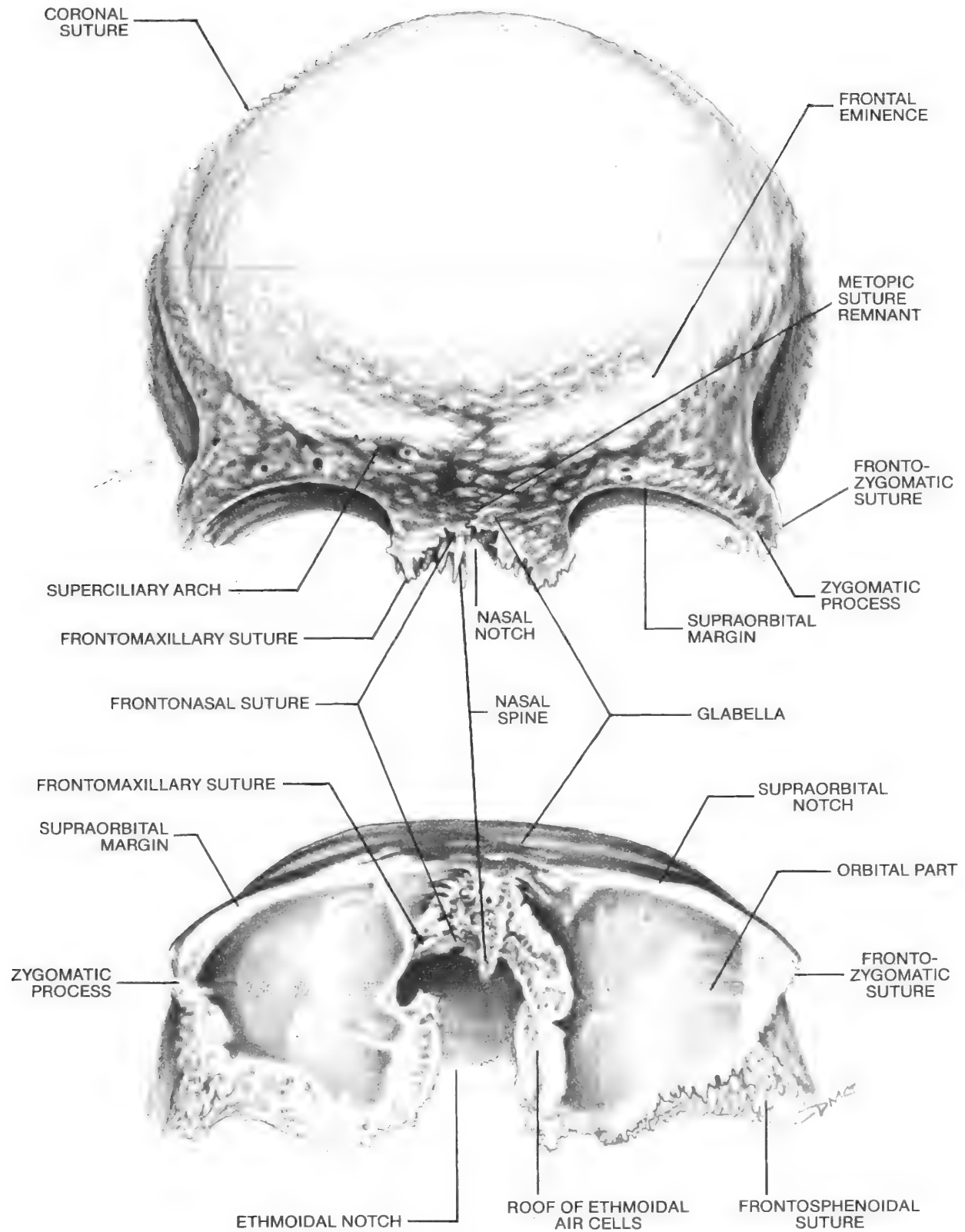
The frontal bone has a general axis of rotation through each of its halves. The general axis of rotation is close to the metopic suture. On sphenobasilar flexion the metopic suture area acts as a hinge and moves slightly posteriorly. The lateral borders of the frontal move laterally.

Articulations

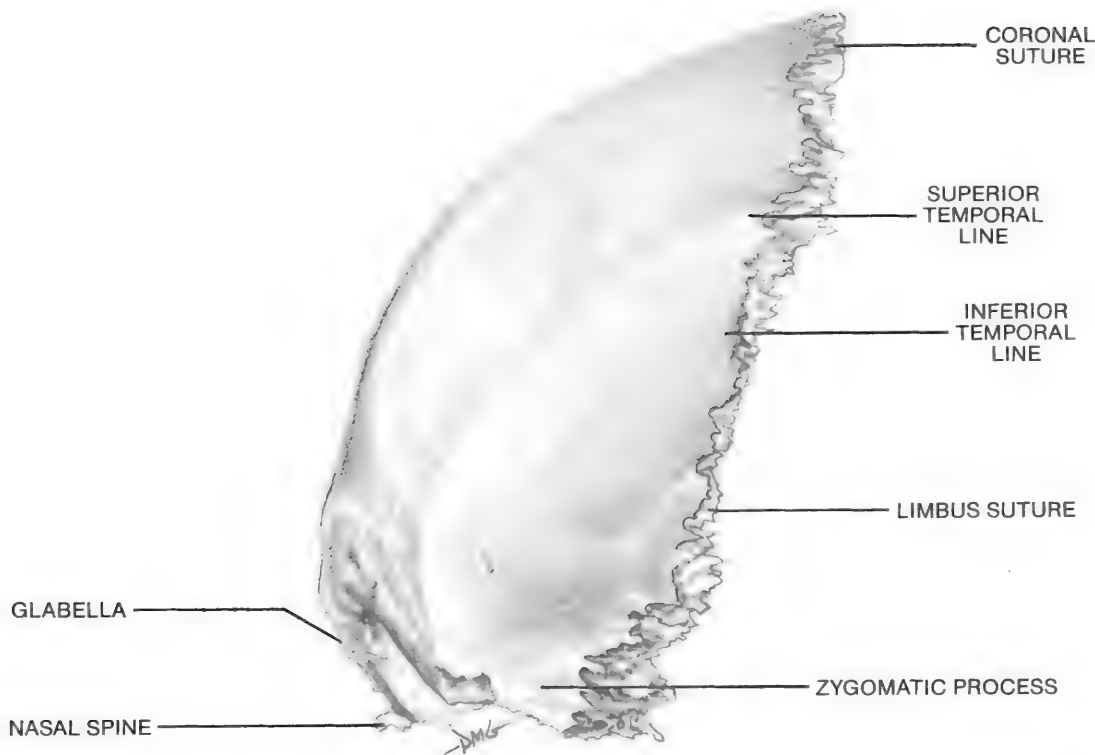
The parietal border is serrated with deep interdigitations to the parietal bone to form the *coronal suture*. The superior aspect of this suture gives an expansion and contraction type of motion. About midway the suture changes more to a limbic suture, with the external bevel on the frontal bone. Along with expansion and contraction at this suture, there is a flaring of the parietal away from the more stable frontal bone. On sphenobasilar flexion, there is a depression of the bregma, the point where the sagittal meets the coronal suture.

Proceeding inferiorly from the pterion, the *fronto-sphenoidal suture* angles anteriorly and is a serrated type, interdigitating with the anterior superior aspect of the greater wing of the sphenoid. The portion of the suture that articulates with the superior aspect of the greater wing of the sphenoid is limbic in nature, with an external bevel on the frontal bone. The

Cranial Osteology



3—36. Frontal bone — anterior and inferior views



3—37. Frontal bone — lateral view

sliding squamous aspect is present but is not as great as that of the squamosal suture. The more serrated portion of the suture associated with the orbital surface is of an expansile nature.

The zygomatic process articulates with the zygomatic bone by a serrated suture. The motion is of expansion and contraction.

The nasal part articulates with the nasal bone, frontal process of the maxilla, and the lacrimal bone. The posterior aspect of the nasal spine articulates with the ethmoid. The ethmoidal notch is occupied by the cribriform plate of the ethmoid bone.

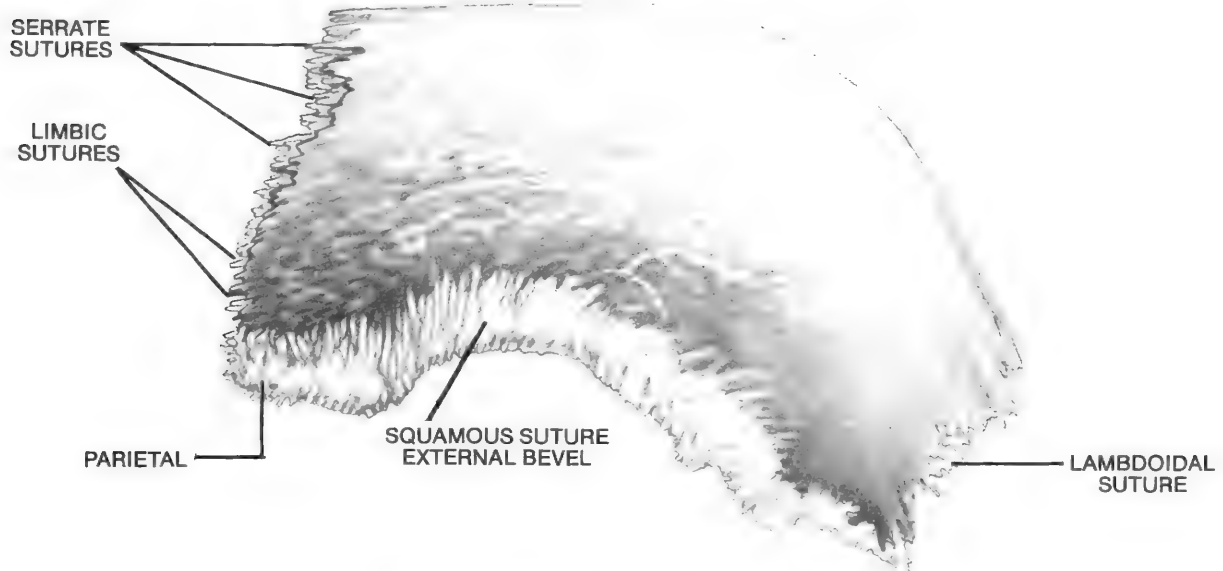
Lateral and posterior to the ethmoidal notch is a serrated suture which articulates with the lesser wing of the sphenoid and has expansion and contraction motion. The tapered points of the lesser wing of the

sphenoid articulate along the posterior borders of the orbital plates.

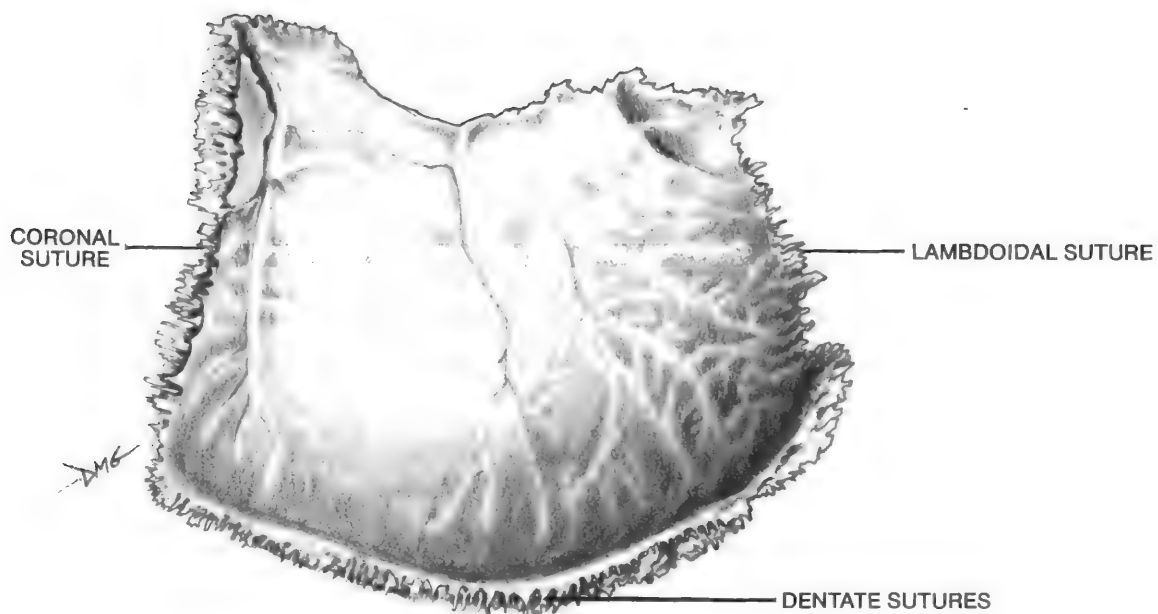
On sphenobasilar flexion (inspiration), the metopic suture area moves posteriorly and the lateral posterior border of the squama moves externally (laterally and anteriorly). This causes the ethmoidal notch to widen, especially in its posterior aspect. The crista galli of the ethmoid moves posteriorly and superiorly, with action of the falx cerebri. The articulation between the greater wing of the sphenoid bone and the frontal bone moves slightly inferiorly, which tilts the superior coronal suture area slightly posteriorly and inferiorly, depressing the bregma. The inferior coronal suture moves slightly anteriorly.

Parietal Bone

The parietal bone forms the side and roof of the cranium. The external surface is convex and smooth, while the internal surface is concave with numerous grooves for the meningeal vessels and depressions for the cerebral convolutions. The superior and inferior temporal lines cross the middle of the bone. The temporal fascia arises from the superior temporal line, and the upper margin of the temporalis is marked by the inferior temporal line.



3—38. Parietal bone - lateral view



3—39. Parietal bone — medial inferior view

Movement

The parietal bone generally moves with an external flaring motion during sphenobasilar flexion (inspiration); that is, the lateral inferior borders move laterally with somewhat of a hinging action at the sagittal suture.

Articulations

The parietal bone has four borders and four angles. The sagittal border articulates with the opposing parietal, forming the *sagittal suture*. The frontal border articulates with the frontal bone, forming half of the *coronal suture*. The point where the sagittal and coronal sutures meet is called the *bregma*, which is equivalent to the anterior fontanelle in the infant. The occipital border articulates with the occipital bone, forming half of the *lambdoidal suture*. The intersection of the sagittal and lambdoidal sutures is called the *lambda*, and it corresponds with the posterior fontanelle in the infant. The point where the lambdoidal suture turns into the *occipitomastoid suture* and joins with the parietal notch is known as the *asterion*. The squamous border is divided into three parts. Articulating with the mastoid portion of the temporal is a thick, serrated section approximately 2 cm long which is called the *parietomastoid suture*. The remaining portion is the squamous border, a significantly beveled external squamous suture. The middle portion is overlapped by the

temporal squama and is called the *squamosal suture*. The anterior portion is overlapped by the superior aspect of the greater wing of the sphenoid and is called the *sphenoparietal suture*.

The section of the parietal bone which articulates with the parietal notch of the temporal bone acts as a pivot for flaring of the temporal squama and the parietal bone on sphenobasilar flexion.

The broad external squamous suture which articulates with the crest of the temporal bone squama and the superior border of the greater wing of the sphenoid allows for internal and external rotation of the bones. This wide bevel provides significant sliding action.

As the coronal suture begins at the pterion, the articulation changes to an internal bevel with some serrated interdigitation with the frontal bone. Approximately the lateral half of the coronal suture is a limbic suture, having the characteristics of a serrated and squamous suture. The internal bevel enables the parietal to move in external rotation to a greater degree than that of the more rigid frontal bone. Progressing medially, the serrated nature of the coronal suture becomes more significant and the motion changes to more of an expansile nature.

The sagittal suture has deep dentate interdigitation, with the motion primarily that of separation and hinge activity.

Maxillary Bone

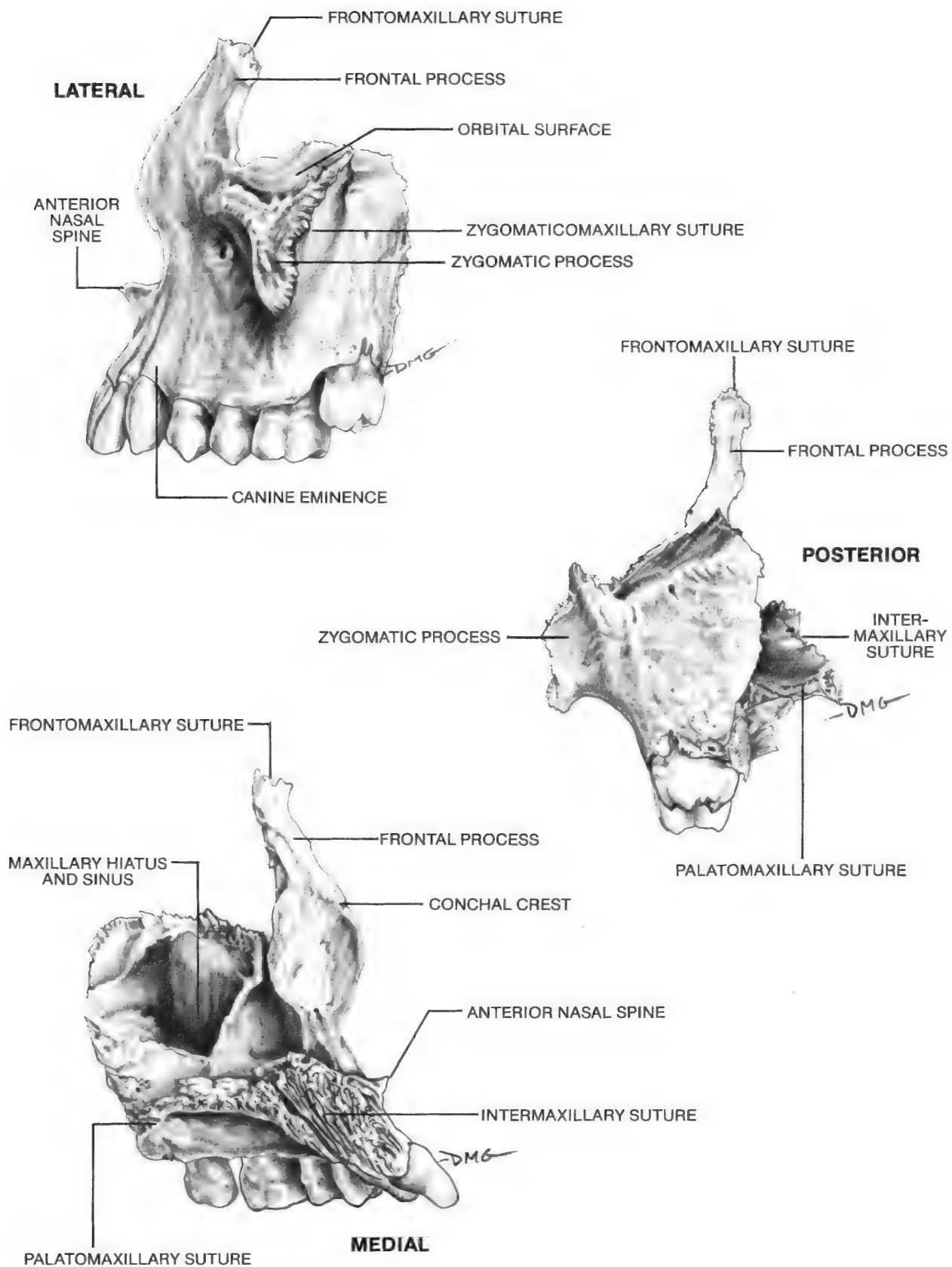


The maxillary bone consists of a body and four processes — the zygomatic, frontal, alveolar, and palatine. The *body*, which contains the maxillary sinus, has four surfaces: the anterior, posterior or infratemporal, superior or orbital, and the medial or nasal.

The anterior or facial surface is directed forward and laterally, and has several elevations corresponding to the roots of the upper teeth. Below the sharp border marking the junction of the anterior orbital surfaces is the infraorbital foramen, which transmits the infraorbital vessels and nerves.

The infratemporal surface is separated from the anterior surface by the *zygomatic process*, from which a ridge extends to the first molar. The *maxillary tuberosity* is posterior to the third molar; it articulates with the pyramidal process of the palatine bone on its medial aspect. Occasionally there is an articulation with the lateral pterygoid plate of the sphenoid bone. From this point originate a few fibers of the medial pterygoid muscle. The pterygopalatine fossa — a groove for the maxillary nerve — is above.

Cranial Osteology



3—40. Lateral, posterior, and medial views of maxillary bone

The superior or *orbital surface* contributes to the greater portion of the floor of the orbit. Medial is the lacrimal notch, and posterior to the notch is an articulation with the lacrimal bone, lamina orbitalis of the ethmoid, and the orbital process of the palatine bone. It forms the anterior margin of the inferior orbital fissure at the posterior. The anterior portion makes up the medial and inferior margins of the eye socket and articulates with the frontal and zygomatic processes. The infraorbital canal begins as a groove at the middle of the posterior border, ending as a foramen below the orbit's margin. The canal yields a branch which runs downward in the anterior wall of the maxillary sinus; it contains the anterior and superior alveolar vessels and the nerve to the front teeth.

The nasal surface opens to the maxillary sinus, which is closed by the ethmoid and lacrimal bones. The medial inferior aspect is a smooth concavity forming the inferior meatus of the nasal cavity. Along the posterior inferior aspect of the maxillary sinus is its articulation with the perpendicular plate of the palatine bone.

The *alveolar process* has deep cavities containing the roots of the teeth. Together with the opposite maxillary bone's alveolar process it comprises the upper dental arch.

The *palatine process* horizontally projects medially from the nasal surface of the maxillary bone. It forms three-fourths of the hard palate, and the superior surface forms a large part of the floor of the nose. The posterior aspect articulates with the palatine bone.

The *frontal process* forms part of the lateral boundary of the nose. The superior border articulates with the frontal bone, and the anterior with the nasal bone.

The *zygomatic process* projects laterally from the angle of separation of the anterior infratemporal

and orbital surfaces, and articulates with the zygomatic bone. It forms part of the anterior facial surface and infratemporal fossa.

Movement

During sphenobasilar flexion (inspiration), the dental arch moves laterally and anteriorly to incline the teeth to a greater degree. The intermaxillary suture moves inferiorly and posteriorly. This action widens the space between the posterior dental arch.

Articulations

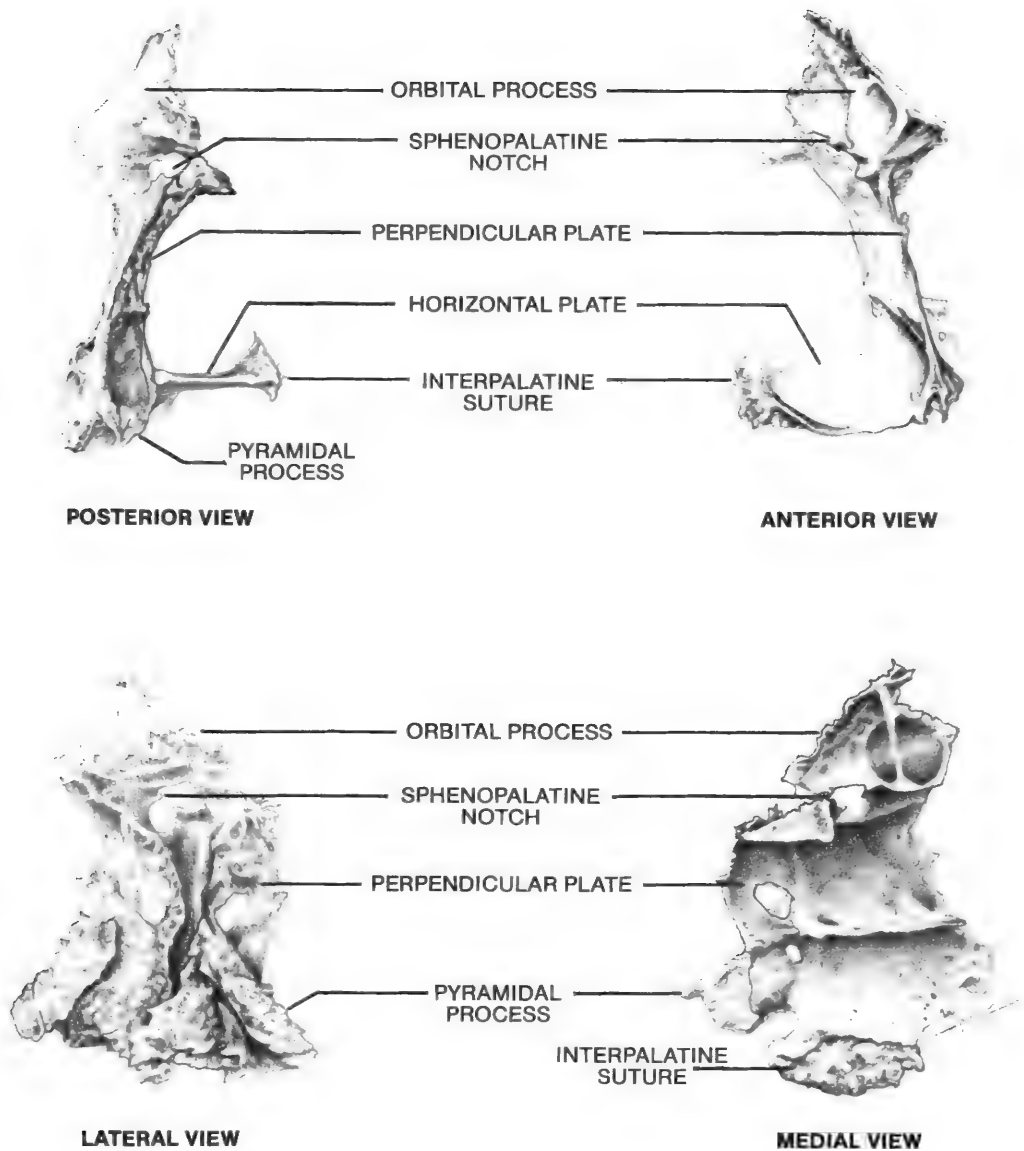
The *intermaxillary suture* has some interdigitation of a serrate nature, but it is primarily a plane suture. The motion is a slight hinge-type action in the posterior and anterosuperior portions of the suture, with slight expansion and contraction at the anterior inferior suture. The *zygomaticomaxillary suture* is a rough, serrated suture facing slightly superiorly, laterally, and anteriorly. The motion between the maxilla and zygomatic bone is primarily that of expansion and contraction. The *frontomaxillary suture* is a serrate suture, providing flexibility for the slight lateral flaring of the dental arch. The junction of the maxillary bone with the ethmoid is a plane suture. The uncinat process of the ethmoid partially closes the maxillary sinus. The *palatomaxillary suture* is lightly serrated in its junction with the horizontal plate of the palatine bone. The articulation with the perpendicular plate of the palatine is a plane suture. The perpendicular plate of the palatine is partly responsible for closing the maxillary sinus posteriorly. The maxillary bone articulates with the lacrimals and nasal concha by plane sutures. The *nasomaxillary suture* is a squamous type with some sliding capability. The vomer articulates into a groove composed of a slight ridge from either maxilla, called the *nasal crest*.

Palatine Bone

The palatine bone is somewhat L-shaped and has two portions: the horizontal and vertical. There are three outstanding processes — the pyramidal, orbital, and sphenoidal.

The *horizontal portion* makes up the posterior one-fourth of the hard palate, and its superior surface forms the back part of the floor of the nasal cavity.

The *vertical part* is thin and oblong. The inferior part is the nasal surface, above which is a crest for articulation with the inferior nasal concha. Above this is a meatus for articulation with the middle nasal concha. The superior meatus is above the ethmoidal crest.



3—41. Palatine bone

The maxillary surface of the vertical part articulates with the maxilla and forms part of the maxillary sinus. A groove between the palatine and maxilla transmits the greater palatine vessels and nerve.

From the superior aspect of the vertical plate arise the orbital and sphenoidal processes, which are separated by the sphenopalatine notch. The *orbital process* is directed superiorly and laterally and articulates with the maxilla, sphenoidal sinus and concha, and the labyrinth of the ethmoid. The orbital surface forms the posterior part of the floor of the orbit, while the lateral surface helps form the inferior

orbital fissure.

The *sphenoidal process* articulates with the base of the pterygoid process and the sphenoidal concha. The posterior border articulates with the medial pterygoid plate.

The *pyramidal process* arises from the junction of the horizontal and vertical plates, projecting laterally and inferiorly to articulate with the pterygoid plates. The grooved intermediate area completes the pterygoid fossa and gives origin to a few fibers of the medial pterygoid muscle.

Movement

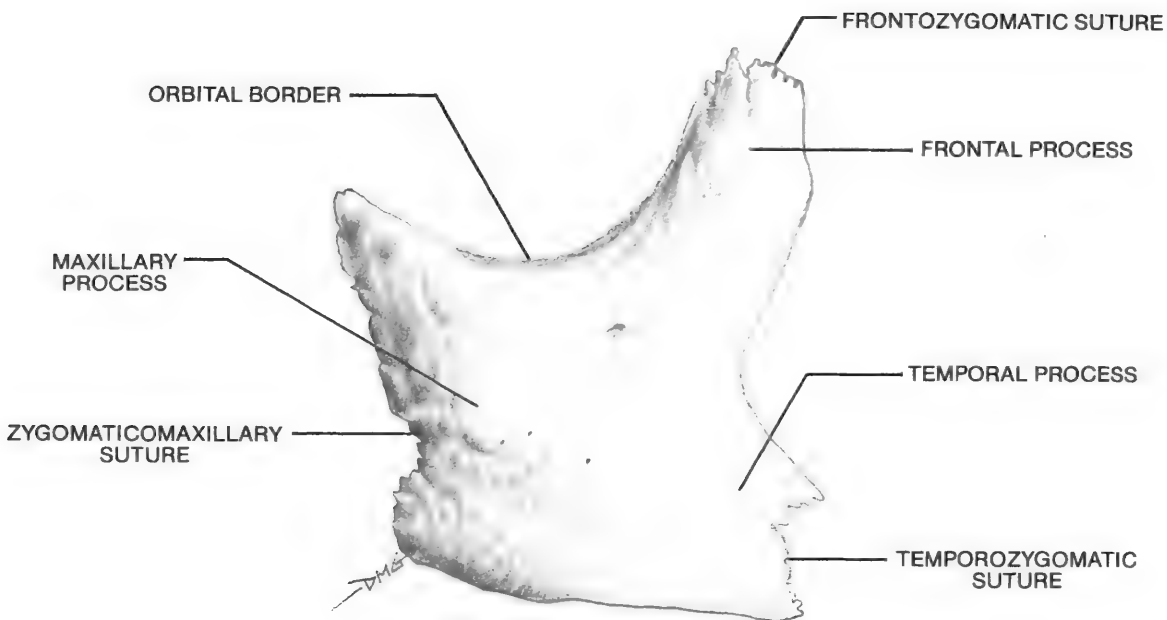
The following motions are with sphenobasilar flexion. The orbital process moves inferiorly with the maxilla and body of the sphenoid. The medial aspect of the horizontal plate moves inferiorly with the palatine process of the maxillary bone. The pyramidal process has plane sutures with the medial and lateral pterygoid plates, and moves posteriorly and

laterally with them. The articulation of the palatine bone with the medial and lateral pterygoid plates is important in the evaluation and correction of certain cranial faults in applied kinesiology. It provides an opportunity to contact the inferior aspect of the sphenoid bone through the oral cavity in various ways.

Zygomatic Bone

The zygomatic bone has three surfaces — the lateral, the orbital, and the temporal — and three processes — the frontal, maxillary, and temporal. It has four borders.

The lateral surface — sometimes called the malar surface — is the external convex surface making up the promontory of the cheek. The temporal surface is concave, facing medially and posteriorly. It presents a surface for articulation with the maxilla. The orbital surface forms most of the anterolateral inferior aspect of the orbit.



3—42. Zygomatic bone — lateral view

Cranial Osteology

The *frontal process* articulates with the zygomatic process of the frontal bone at its anterior superior aspect, and with the greater wing of the sphenoid at its posterior inferior aspect. The *maxillary process* is sometimes not considered as a process, but rather as the maxillary border. It is a triangular-shaped, roughened area which articulates with the zygomatic process of the maxilla. The *temporal process* is a long, bar-like process directed posteriorly to articulate with the zygomatic process of the temporal bone.

The posterior border curves gently and is continuous with the temporal line of the frontal bone and the zygomatic arch of the temporal bone. It affords attachment for the temporal fascia. The posterior inferior border is an attachment for the masseter muscle. The anterior medial border forms part of the rim of the eye socket, and the short

medial border forms the anterior boundary of the inferior orbital fissure.

Movement

The zygomatic bone moves with the sphenoid, temporal, and maxilla. The zygomatic bone basically rides superiorly, laterally, and anteriorly with the maxilla.

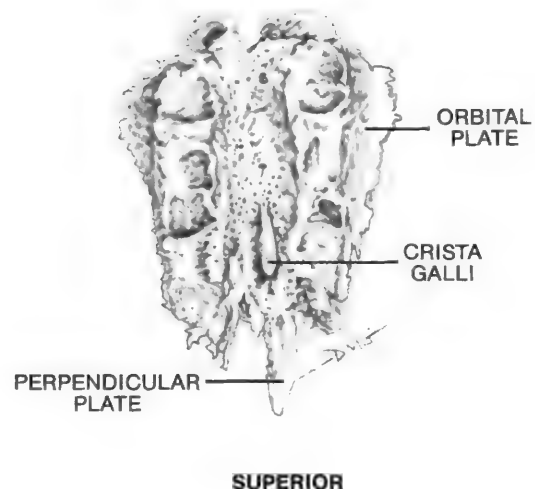
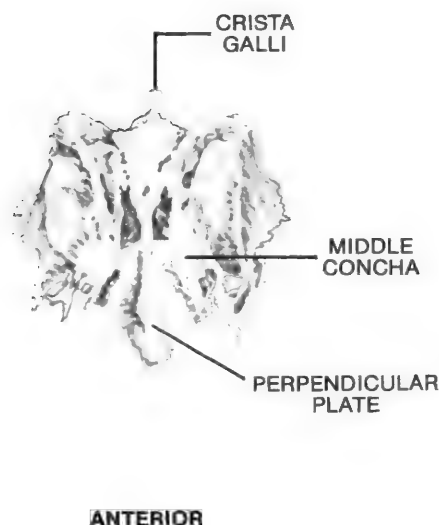
Articulations

The *zygomaticomaxillary suture* is a rough, serrated suture triangular in shape. Its motion is primarily expansion and contraction. The *sphenozygomatic suture* is limbus, with the external bevel on the sphenoid. The *frontozygomatic* and the *temporozygomatic* are both serrate sutures, which primarily expand and contract.

Ethmoid Bone

The ethmoid bone is exceedingly light, with many of its portions in the dried bone similar to parchment paper. It consists of four parts: the horizontal portion called the cribriform plate, which forms part of the anterior base of the cranium; a vertical part, called the perpendicular plate; and two lateral masses called labyrinths.

The *cribriform plate* fills the ethmoidal notch of the frontal bone. The *crista galli*, an attachment for the falx cerebri, rises from its middle. The cribriform plate is perforated by many olfactory foramina for the olfactory nerves.



3—43. Ethmoid bone

The *perpendicular plate* is very thin. It descends in the midline from the cribriform plate and forms the upper part of the nasal septum. The anterior border articulates with the spine of the frontal bone and the crests of the nasal bones. The posterior border articulates above with the sphenoidal crest and below with the vomer.

The *labyrinth* consists of a number of thin-walled, ethmoidal air cells.

Movement

On sphenobasilar flexion, the crista galli moves posteriorly and superiorly with the attachment of the falx cerebri. The posterior aspect of the cribriform plate lowers with the anterior aspect of the body of the sphenoid.

Articulations

The ethmoid articulates with thirteen bones. The *frontoethmoid suture* is a plane suture between the lateral aspects of the cribriform plate, with the margins lateral to the ethmoidal notch of the frontal bone. There is communication from the frontal sinuses to the labyrinth, especially at the anterior. The posterior border of the perpendicular plate articulates with the sphenoidal crest above and with the vomer below. The other bones with which the ethmoid articulates are the two nasals, two maxillae, two lacrimals, two palatines, and two inferior nasal conchae.

Vomer Bone

The vomer is a thin, flat bone forming the posterior and inferior parts of the nasal septum. It is so named because of its resemblance to a plowshare. The bone is not entirely flat; it thickens on the posterior superior aspect to form two wings which make a deep furrow articulating with the rostrum of the sphenoid.

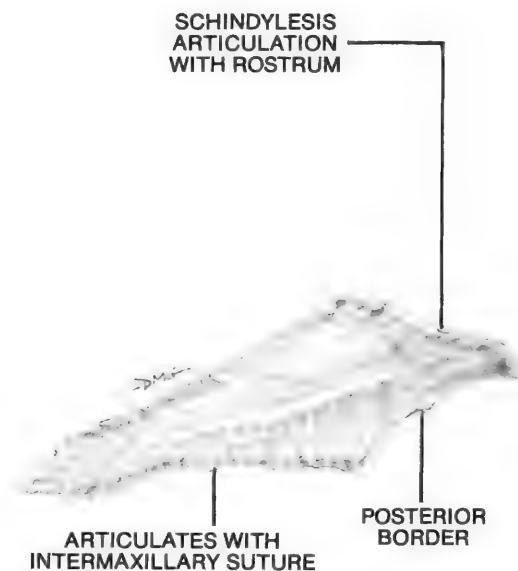
Articulations

The lateral aspects of the wings, or alae, articulate with the vaginal processes of the medial pterygoid plates of the sphenoid and with the sphenoidal processes of the palatine bones. The inferior border of the vomer articulates into a crest along the intermaxillary and interpalatine sutures. The superior aspect of the anterior border articulates with the perpendicular plate of the ethmoid; the posterior border is free.

Movement

The posterior inferior aspect of the vomer, which articulates in the midline of the cruciate suture, moves inferiorly with the palatine process of the maxillary bone and the horizontal plate of the palatine bone on sphenobasilar flexion. The posterior superior aspect, articulating with the rostrum of the sphenoid, moves inferiorly.

The vomer is a connecting structure between the hard palate and the sphenoid, important for certain applied kinesiology challenges in examination. It also transmits therapeutic forces.

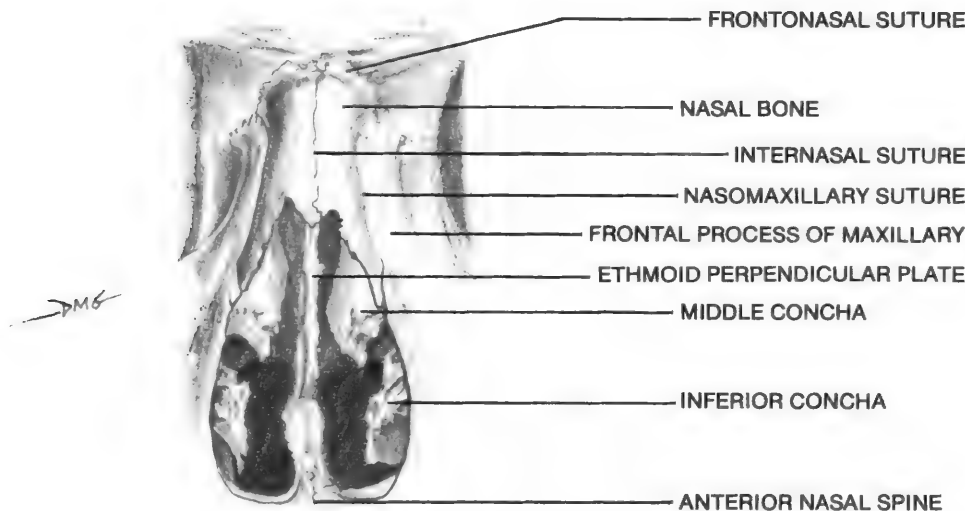


3—44. Vomer bone — lateral view

Nasal Bone

The nasal bones are small linear bones which comprise the bridge of the nose. They have two surfaces and four borders. The external surface is convex from side to side; from above down it is mostly concave, and at the lower area becomes convex. The superior border is serrated to make the frontonasal suture. The lateral border articulates with the frontal process of the maxillary bone,

forming the nasomaxillary suture. It is a plane suture, closely resembling a schindylesis with its somewhat tongue-in-groove nature. The medial border is plane, forming the internasal suture. The inferior border is thin, notched, and continuous with the nasal cartilage. The internal surface is grooved from the superior to inferior border, where the ethmoidal nerve lies.



3—45. Anterior view into the nasal cavity.

Inferior Nasal Concha

The inferior nasal concha is a curved layer of bone lying horizontally in the lateral wall of the nasal cavity, at approximately the level of the inferior orbital margin. Observing the concha from the anterior end by looking through the nasal opening, it appears somewhat scroll-like and has a convexity arising from its superior lateral surface. The lateral surface is the articulating one, articulating with four bones. The medial surface is convex, with numerous apertures and grooves for vessels. The lateral surface is concave and forms part of the inferior meatus. The superior border is thin, irregular, and divided into three parts. The anterior portion articu-

lates with the conchal crest of the maxilla, and the posterior portion with the conchal crest of the palatine. The middle portion is divided into three processes. The lacrimal process is small and pointed; it articulates with the lacrimal bone and with a groove on the back of the frontal process of the maxilla, forming the canal for the nasolacrimal duct. The ethmoidal process is posterior to the lacrimal process and articulates with the ethmoid. Finally, the mammillary process articulates with the maxilla, forming a part of the medial wall of the maxillary sinus.

Movement

The movement of air on inspiration may have some influence on cranial bone motion. It has been observed by Goodheart¹³ that in some instances an individual does not have the option to breathe, either through the nose or the mouth, without causing apparent dysfunction, indicated when a strong indicator muscle weakens on manual muscle testing. It seems obvious that the inferior conchae are moved by the bones with which they articulate. It is unclear whether the passage of air influences the conchae in

such a manner that they in turn influence the bones with which they articulate.

Articulations

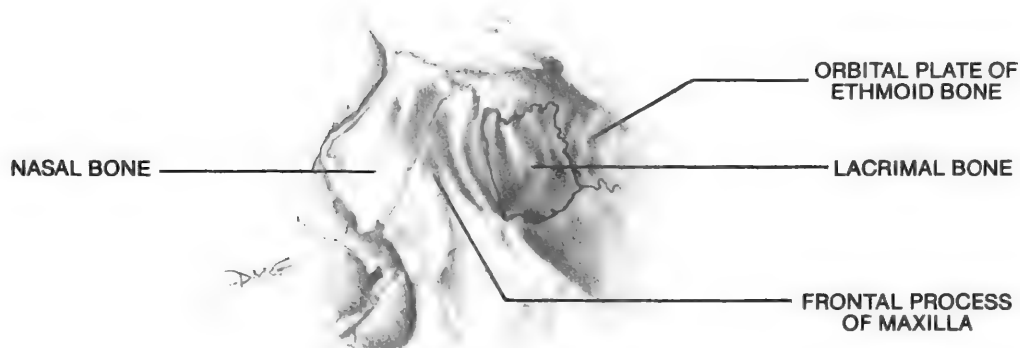
The inferior nasal conchae articulate by plane sutures with four bones — the ethmoid, maxilla, lacrimal, and palatine. Their exact movements and significance are unclear, inasmuch as the articulations cannot be influenced directly or indirectly by the applied kinesiology method of restricting motion (see page 87).

Lacrimal Bone

The lacrimal is the smallest facial bone; it is very thin and fragile, much like parchment paper. It has two surfaces and four borders. Its lateral or orbital surface has a vertical ridge called the posterior lacrimal crest. Anterior to this crest is the lacrimal sulcus which, with the frontal process of the maxillary bone, makes up the lacrimal fossa housing the lacrimal sac and nasolacrimal duct. Posterior to the posterior lacrimal crest is the surface which forms part of the orbit. On the medial or nasal surface there is a furrow formed as a result of the posterior lacrimal crest. Anterior to the furrow, the

bone forms part of the middle meatus of the nose. Posterior to the furrow, the medial surface articulates with the ethmoid, completing some of the anterior ethmoidal cells.

The superior border articulates with the frontal bone, the posterior border with the ethmoid, and the anterior border with the maxilla. The inferior border is divided into two sections, the posterior part of which articulates with the maxilla. The anterior part descends to articulate with the lacrimal process of the inferior nasal concha; it assists in forming the canal of the nasolacrimal duct.



3—46. *Anterolateral view of orbital and nasal areas.*

Meninges

Much emphasis has been placed on the meninges, especially the dura mater, in relation to cranial motion.^{6, 7, 12, 23, 36} The dura mater is a tough, shiny, inelastic membrane enveloping the brain. An extension of the cerebral dura mater envelops the spinal cord and attaches to the sacrum; it will be considered later when pelvic and cranial motion are correlated.

The cerebral dura mater lines the interior of the skull, separating the brain into various compartments by several folds. These folds, along with the cerebrospinal fluid, cushion and protect the brain. The external surface of the dura mater adheres to the cranial bones, acting as an internal periosteum. This is called the endosteal layer; it is continuous through the sutures and foramina of the skull with the external periosteum. The inner layer of dura is called the meningeal layer and is closely united with the endosteal layer. The meningeal layer projects through the foramina with the cranial nerves and becomes fused with the epineurium of the nerves. The meningeal layer projects four folds, forming four compartments as previously mentioned.

Tentorium Cerebelli

The tentorium cerebelli is a tent-like fold covering the cerebellum and supporting the occipital lobes of the cerebrum. Posteriorly and posterolaterally, it attaches to the bony lips of the occipital bone which

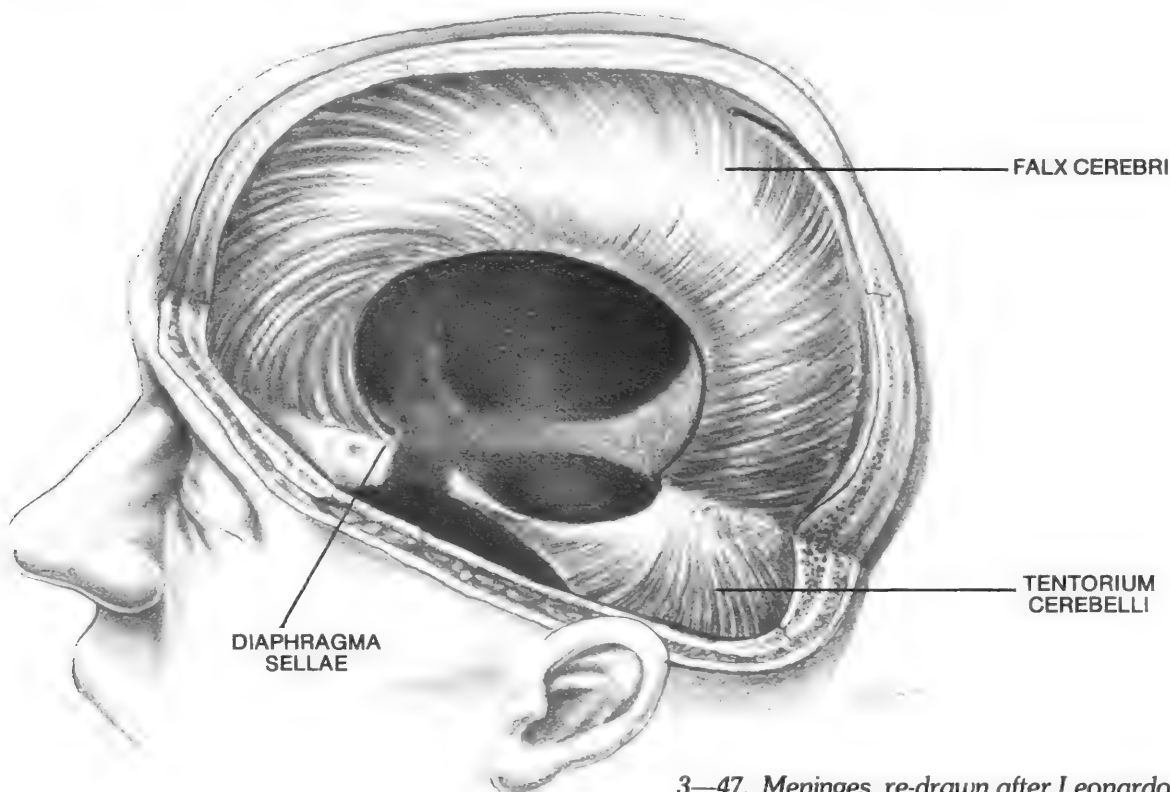
borders the transverse sinus. At the lateral aspect of the transverse sinus, the attachment changes to the superior ridge of the petrous portion of the temporal bone. From the apex of the petrous portion, it attaches to the lateral border of the clivus, up to the posterior clinoid process of the sphenoid bone. The medial free border runs from the anterior clinoid process to the continuous attachment with the falx cerebri, leaving a large oval opening — the tentorial incisura — for the midbrain and the anterior part of the superior surface of the vermis of the cerebellum.

Falx Cerebri

The falx cerebri is continuous superiorly from the tentorium cerebelli. It attaches posteriorly to the internal occipital protuberance, running superiorly to the sagittal suture and then to the frontal bone, and finally ending on the crista galli of the ethmoid bone. The superior sagittal sinus runs along this entire length. The lower margin is free and concave, and contains the inferior sagittal sinus.

Falx Cerebelli

The falx cerebelli is continuous inferiorly from the center of the tentorium cerebelli. Its posterior border is attached to the internal occipital crest and contains the occipital sinus. The free border projects anteriorly into the posterior cerebellar notch.



3—47. Meninges, re-drawn after Leonardo da Vinci.

Diaphragma Sellae

The diaphragma sellae connects the clinoid attachments of the two sides of the tentorium cerebelli. It forms a roof over the sella turcica and almost completely covers the hypophysis, leaving a small opening in the center for the infundibulum.

General Discussion

The hypothesis of the dura mater's importance in cranial motion originated with Sutherland.³⁶ He called the mechanism the "... reciprocal tension membrane," consisting of the falx cerebri and the tentorium cerebelli. Their function is to cause "... movement of the articulations and at the same time regulating or limiting the normal range of articular mobility." The attachments of the falx cerebri and the tentorium cerebelli are referred to as poles of attachment. The falx cerebri has an anterior superior pole of attachment on the crista galli, and the tentorium cerebelli has an anterior inferior pole of attachment on the clinoid processes of the sphenoid bone. The tentorium attachment to the superior ridge of the petrous portion is considered the lateral pole, with the posterior pole at the occipital attachment.

The concept of the reciprocal tension membrane considers the common origin of the bilateral tentorium cerebelli and falx cerebri to be at their junction where the straight sinus is located. This area is called "the Sutherland fulcrum."²³ The secondary insertion is at the poles described.

Page²⁷ points out the continuity of fascia throughout the body. There is continuity of the fascia from the apex of the diaphragm to the base of the skull. Not only does it extend to the outer surface of the sphenoid, occipital, and temporal bones; it enters the foramina in the base of the skull around vessels and nerves to join the dura mater. Thus the motions of thoracic respiration have continuity directly with the dura, as well as that described in Chapter 7 with the pelvis.

Although there have been various attempts to describe the motion of the reciprocal tension membrane,^{6, 7, 21, 23, 30, 36} there is discrepancy in thought by most authorities and many students about the cranial primary respiratory mechanism. This is reasonable because of the inaccessibility of these structures in the living human. Even entering into the system on laboratory animals will result in questionable observations because of changes to the normally functioning mechanism. It seems reasonable to avoid the controversy regarding the exact motions of the reciprocal tension membrane; thus an additional hypothesis about an already very clouded subject will not be attempted. It is important, however, to further discuss some of the general characteristics of the

probable mechanisms of cranial nerve entrapment, interference with cerebrospinal fluid pressure control and movement, and possible influence on blood vascular circulation to the cranium.

Clinical evidence indicates that there are many possible entrapments of cranial nerves from various types of cranial faults. The evidence includes immediate improved function of muscles which are supplied by cranial nerves as observed by applied kinesiology testing. These include the extrinsic ocular muscles, muscles of mastication, hyoid muscles, facial muscles, sternocleidomastoid, and upper trapezius. Other, less definitive, clinical improvements that correlate with cranial nerve function are often seen immediately following cranial fault correction. Although it is difficult to deny these clinical changes, it is mandatory for further development in this field that electrophysiologic studies be done to determine the exact mechanism of the improvement. (The possible mechanisms of entrapment will be discussed later under cranial nerves.)

Cerebrospinal fluid is an important consideration in the cranial concept which Sutherland developed.³⁶ Included in his hypothesis is that the brain has an involuntary rhythmic movement which includes dilation and contraction of the ventricles. This movement causes cerebrospinal fluid circulatory activity, which moves the arachnoid and dural membranes and affects mobility in the basilar articulations through the reciprocal tension membrane.

Fluctuation of cerebrospinal fluid as described by Sutherland is an important part of the osteopathic concept of examination and treatment of the cranial primary respiratory mechanism. The concept is to direct cerebrospinal fluid motion to a suspected area of restricted articular motion. This is accomplished by directing slight pressure to the pole opposite that to be examined by palpation. For example, slight pressure is placed on the left frontal bone to direct fluid to the right lambdoidal suture. It is expected that motion at the lambdoidal suture will increase if there is normal function. The fluid fluctuation is directed in this manner rather than following the general principles of hydrodynamics because it is held to be a mechanism of the reciprocal tension membrane.

Standard physiology acknowledges no regulation of cerebrospinal fluid production, absorption, or pressure control in relation to the primary respiratory mechanism. The quantity of cerebrospinal fluid is considered approximately 9% of the total volume of the cavity enclosing the brain and spinal cord. Daily production is approximately 800 ml, which is five to six times as much as the total volume of fluid in the entire cerebrospinal fluid cavity.⁸ The fluid is secreted by the choroid plexuses located in all the ventricles,

Cranial Osteology

with additional fluid secreted by all the ependymal surfaces of the ventricles. Most of the fluid is produced in the two lateral ventricles. It flows through the foramen of Monro to the third ventricle; with the additional fluid from there it flows through the aqueduct of Sylvius to the fourth ventricle. There are three outlets from the fourth ventricle — the two lateral foramina of Luschka and the foramen of Magendie — which enter the cisterna magna lying behind the medulla and beneath the cerebellum. The cisterna magna is continuous with the subarachnoid space, surrounding the entire brain and spinal cord. Before reaching the cerebrum it must flow through the small tentorial opening around the mesencephalon, where the flow is sometimes impeded.

Loss of cerebrospinal fluid is primarily through the arachnoid villi, which are berry-like tufts of arachnoid protruding into the venous sinuses. Through these bodies the cerebrospinal fluid — including large particles such as protein molecules — empties directly into the venous blood. The escape of cerebrospinal fluid along the roots of the spinal nerves has been demonstrated.^{10, 20} This was questioned by Somberg,³³ but his work was critically analyzed as incorrect by Hassin.¹⁹

Possible influence of the cranial primary respiratory mechanism on blood vascular function has been discussed on page 22. A brief correlation here will suffice.

The influence of cerebrospinal fluid on blood circulation is a clinical observation. Often when idiopathic hypertension is present, it is brought under control by the correction of cranial faults. The reduction of both systolic and diastolic blood pressure in these cases is often observed as rapidly as the

physician can re-test the blood pressure after cranial correction. The hypothesis is that cerebrospinal fluid pressure is low because of cranial dysfunction. The body raises the blood pressure, which expands the venous system in the cranium and spinal cord; thus the cerebrospinal fluid pressure increases. Correction of the cranial fault returns the cerebrospinal fluid pressure to normal, reducing the necessity for the increased blood pressure to elevate the cerebrospinal fluid pressure. This is a working hypothesis which has not been evaluated with controlled studies; however, it seems clinically sound.

It is possible that there is some involvement in blood pressure regulation by way of the cranial primary respiratory mechanism as a result of peripheral entrapment of the glossopharyngeal nerve supplying the carotid sinus. The carotid sinus is not a regulator of blood pressure over prolonged periods, since it adapts to continuous high or low blood pressure over several days.¹⁸ There is the possibility, however, that the characteristic of the nerve impulse may be changed as the result of cranial faults by way of entrapment of the glossopharyngeal nerve.

Blood circulation to the brain is known to be extremely important in normal function. Other than clinical observation, there have been no studies indicating any influence of the cranial faults on this circulation. It seems probable that there is some influence because of the apparent improvement of cerebration following cranial corrections. The symptomatic complex present prior to correction is very similar to an individual's thinking ability when in a low blood sugar state. The increased "clear-headed" improvement in thinking often results immediately after cranial correction.

Integration of Cranial Movement

There is minimal movement between the articulations of the skull. Total skull motion is magnified by the number of articulating surfaces. The skull is made up of twenty-two bones, with well over one hundred articulating surfaces. Its motion appears to be the result of (1) an inherent motion of the brain and cerebrospinal fluid;^{23, 36} (2) intrinsic muscles of the skull, primarily those of mastication; (3) extrinsic muscles of the skull; (4) thoracic respiration; and (5) pelvic motion.

This discussion will attempt to put the anatomy

previously presented in the hypotheses of the "cranial primary respiratory system" into perspective. Some of the hypotheses presented here refer to extrinsic factors as mobilizers of the skull; these are important autocorrection mechanisms when movement fails because of dysfunction of the cranial primary respiratory mechanism Sutherland³⁶ described.

Also presented in this discussion is a brief description of cranial embryology and an integration of the movement of the cranial bones.

Embryology

The skull begins to develop at the end of the first month as a thickening of the mesenchyme, which is called the *desmocranium*. During the second month, the *chondocranium* develops; this is the stage wherein some of the membranous structures change to a cartilaginous stage. Not all the skull bones develop at this stage; primarily it is the bones of the base of the cranium which develop in cartilage. These are the occipital bone (except the squama between the parietals); the petrous portion of the temporal bones; the body, lesser wings and roots of the greater wings of the sphenoid; and the ethmoid. The cranial bones which are formed in membrane (mesenchymatous) are the parietals, the frontal bone, the squamous portion of the temporal bone, and the occipital squama between the parietals. Thus the entire base of the cranium, with the exception of the orbital plates of the frontal and the lateral parts of the greater sphenoidal wings, are preformed in cartilage; the rest of the vault is ossified directly from membrane.

The *viscerocranium* is derived from the *branchial arches* or *visceral arches*. The branchial arches may provide a very important correlation between thoracic respiration and the cranial primary respiratory mechanism. These are six paired arches which develop on the lateral aspects of the head in the vicinity of the hindbrain. These structures are the formation of the gill-arches of fish and in some amphibians. It is through these gill slits that respiratory water flows.¹ In the human, the branchial apparatus is responsible for the formation of the mandibular region and the entire neck.

It is interesting that in 1899 Sutherland made an observation about the "... beveled articular surfaces of the sphenoid bone. Suddenly there came a thought — I call a guiding thought — 'beveled, like the gills of a fish, indicating articular mobility for a respiratory mechanism.'"³⁵ A comparison of embryologic anatomy with the branchial region being the origin of the gill mechanism in fish and amphibians, and also the origin of structures which appear to influence cranial motion, is interesting to relate to Sutherland's observation of the sphenoid bone. Of course, the sphenoid does not develop from the branchial arches; however, his observation is an interesting correlation with the history of knowledge of cranial function.

This correlative embryologic anatomy may give some insight to the parallel activity of thoracic respiration and the cranial primary respiratory mechanism. Further study in this area can be enhanced by noting the admonition in *Gray's Anatomy* (British edition) in discussion of the development of individual systems within embry-

ology. It states that it is "... customary and convenient to limit attention to individual systems in their further development; but it must never be overlooked that the analysis of the whole organism into such divisions — however attractive on morphological and functional grounds — is largely a product of the sequential nature of human perception. Not only do several systems into which we divide the organism (for study) develop simultaneously, they also interact and modify each other."³⁷ This admonition to consider all parts and systems as integrated should be applied to both the study of embryology and also that of total health, as applied kinesiology attempts to do. With this thought in mind, study a comparison of the structures which develop from the branchial apparatus in gill-bearing organisms and in the human.

The gills develop between the branchial arches, which are a series of bars. In the human, the first pair of branchial arches is called the mandibular arches. These form the gnathostomata, or the upper and lower jaws. The second arch — the post-mandibular — is the hyoid arch which forms the varied hyoid elements. In bony fish, the hyoid arch contributes to the formation of a gill cover; the remaining arches persist as the supports of the gill apparatus.

In the human, the mandibular and hyoid arches represent the first and second branchial arches respectively. They are situated between the primitive mouth and the cardiac swelling. The third and fourth arches are depressed by the overlapping of the hyoid arch, while the fifth and sixth arches are not recognizable externally. Some authorities recognize only five arches.¹ The arches below the hyoid arch contribute to the formation of many important structures, such as the pharynx, epiglottis, parathyroid, part of the tongue, etc. The branchial arches are composed of ectoderm and endoderm, separated by a mesenchymal core. The mesenchyme develops various muscles, bones, and blood vessels. *Gray's Anatomy* (British edition)³⁷ states that with the exception of the bone development, "... most of the remainder of the core of mesenchyme becomes striated muscle, which usually migrates and may lose all connection with the skeletal elements in arches which cease to carry out their original respiratory function. The identities of these muscle masses, where they assume new functions, can nevertheless be traced by reference to their nerve supply." Some of these muscles include those of mastication, anterior and posterior bellies of the digastric, epicranial, muscles of expression, auricular, etc.¹ This correlation in comparative anatomy of the gill-bearing vertebrates and the human seems to neurologically tie in the respiratory mechanism of the fish and amphibian with the influence of thoracic respira-

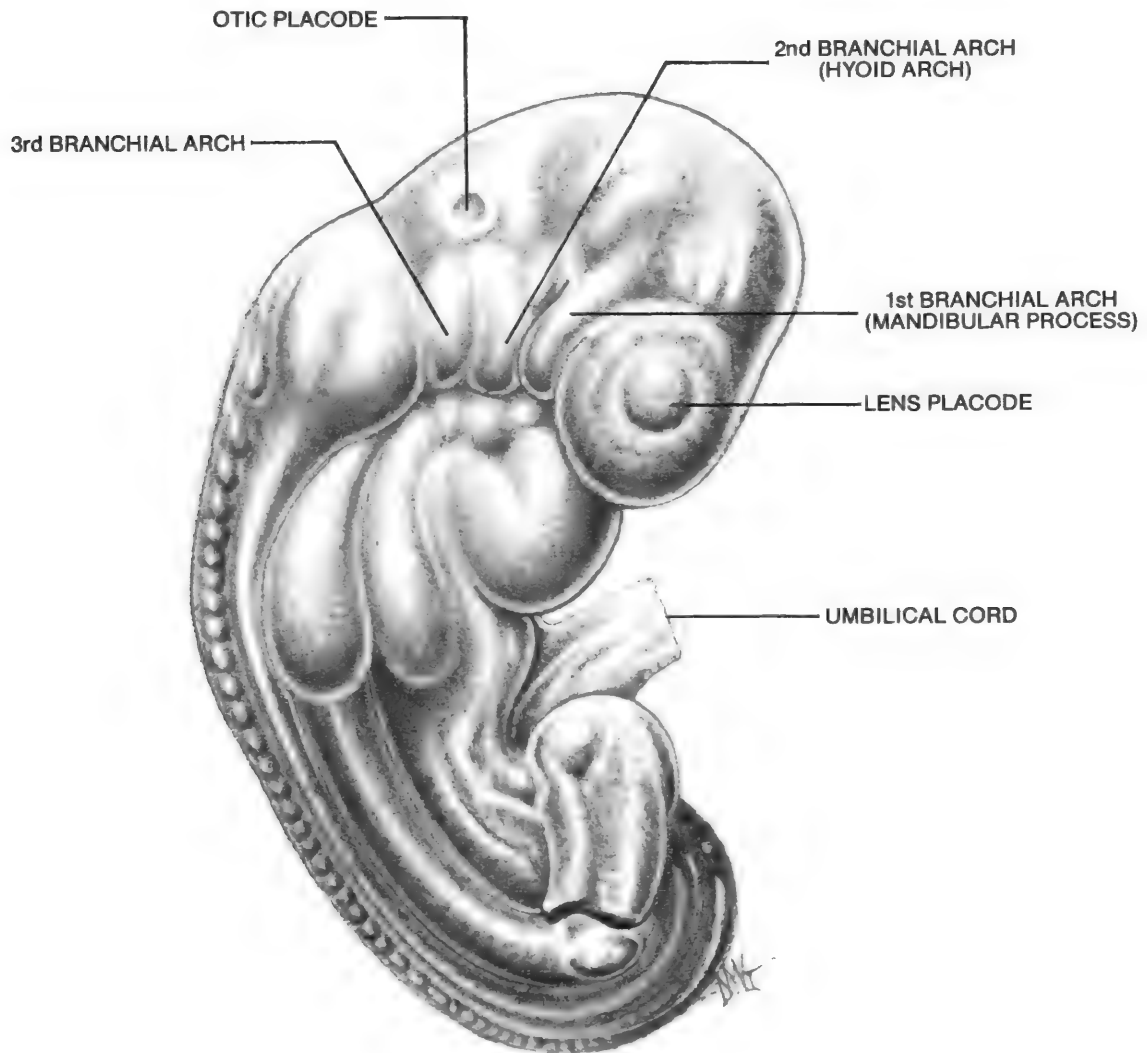
Cranial Osteology

tion on the cranium in lung-bearing animals. Gray's *Anatomy* (British edition)³⁷ states that regardless of the distance that a muscle mass migrates from its site of development, the original innervation nearly always persists.

A step toward the mammalian breathing apparatus is that of the modern amphibians. Their method of breathing is by buccopharyngeal movements. This movement is accomplished by depressing the floor of the mouth, thus increasing the volume of the buccal cavity. With the cavity full of air, the external nares are closed and the buccal floor is elevated, thus

forcing air into the lungs.³² As will be seen later in this text, there is a significant correlation between the mandible and its muscular function with the cranial primary respiratory mechanism. There is specific motion of the mandible with cranial movement, as well as its activity in mastication enhancing or disturbing cranial motion.

Direct investigations of comparative anatomy *in vivo* of the cranial-sacral primary respiratory system may provide increased knowledge to further enhance therapeutic efforts directed toward function in this highly important area.



3—48. Twenty-six day old human embryo.

Correlation of Skull Movement

Skull Movement

Skull movement will be discussed generally according to the manner in which it develops. First the cranial vault will be discussed, broken down into the basilar part and the vault bones. Most of the basilar part is formed in cartilage and is the basis for movement with all other structures. The vault bones, which are formed in membrane, will then be discussed. After the motion of the cranial vault has been completed, facial bone movement will be discussed. The entire discussion will have some repetition as far as the motions described with the anatomy of each bone. This is necessary to tie together the overall general motion of the skull.

Cranial Vault

The basilar portion of the cranial vault is made up of the sphenoid bone; the occipital bone, except for the upper portion of its squama; the petrous portion of the temporal bone; the orbital plates of the frontal bone; and the ethmoid. The only portions of the base of the skull not preformed in cartilage are the orbital plates of the frontal bone and the lateral parts of the sphenoid's greater wings.

The basic motion of the skull can best be understood by first considering the motion of the sphenoid and its relation to the occipital bone. The sphenoid appears to be the key bone in cranial motion. It articulates with twelve other bones, which in turn provide a link to the entire mechanism.

The terms "flexion" and "extension" in cranial terminology refer to movement of the bones in the sagittal plane or about a coronal axis. Flexion, as usual, refers to bending or increasing the angle of an articulation; extension refers to decreasing the bend at the articulation. Rotation refers to movement of the bone in the coronal plane or about a sagittal axis, with external rotation being away from the midline.

The terms "sphenobasilar flexion" and "inspiration" are used to denote the same motion in cranial terminology. Sometimes the term "inspiration" refers to the cranial primary respiratory mechanism movement of sphenobasilar flexion, and sometimes it indicates thoracic inspiration. In this text, when the term "inspiration" refers to a voluntary act of the patient or subject, thoracic respiration is indicated. When inspiration refers to the cranial primary respiratory mechanism, it will be in conjunction with motion of the cranium as sphenobasilar flexion. The terms "expiration" and "sphenobasilar extension" have a similar reference to that of inspiration and sphenobasilar flexion.

The skull motion that will be emphasized throughout this discussion is that of sphenobasilar

flexion, which is inspiration of both the skull and thoracic inspiration if the two are synchronized. Remember that the cranial primary respiratory mechanism is autonomous and continues when the breath is held, or if thoracic respiration is extremely rapid or slow.

Flexion of the sphenobasilar junction is movement of the sphenoid's clivus and the occipital bone's basilar portion superiorly and very slightly posteriorly. The general axis of rotation of the sphenoid is anterior and slightly inferior to the sella turcica in alignment with the sphenosquamous pivot. The anterior body of the sphenoid moves inferiorly and the pterygoid processes move posteriorly and slightly laterally and inferiorly.

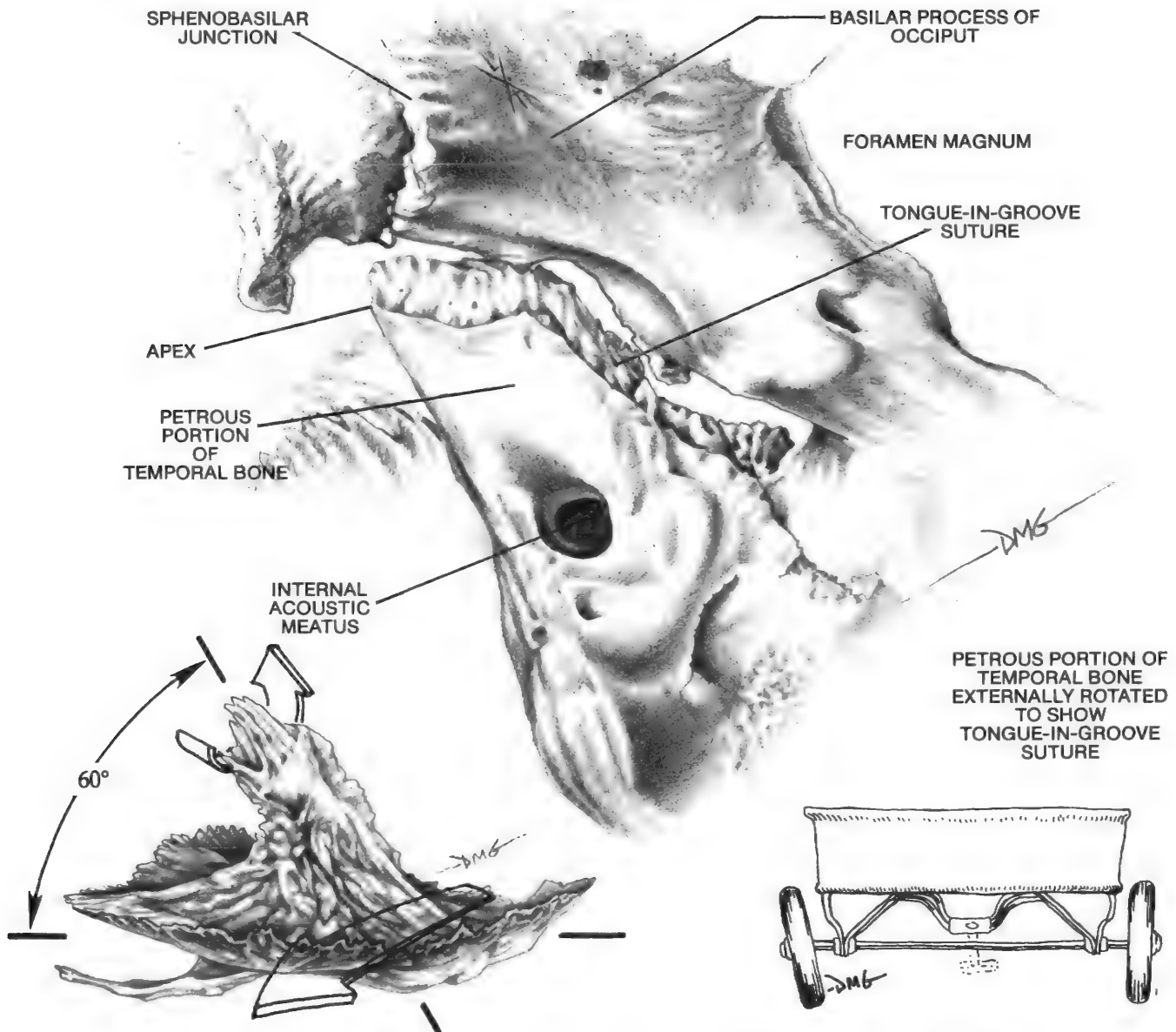
The occipital bone's general axis of rotation — superior to the foramen magnum at the general height of the sphenobasilar junction — is less specific than that of the sphenoid. Thus on sphenobasilar flexion, the foramen magnum moves anteriorly while the superior aspect of the squama moves posteriorly.

It is important to understand the motion at the sphenobasilar junction because it provides a basis for understanding the continuity of cranial motion. An interlink in the movement of the sphenoid and occipital bones is the temporal bone, which provides a very important lever for correcting improper functioning of the sphenoid and occipital bones.

The temporal bone can be considered by sections in its movement with the sphenoid and occipital bones. The apex of the petrous portion articulates along the lateral portion of the basilar portion of the occipital bone by a tongue-in-groove articulation. The same type of articulation is present posterior to the jugular process. As the basilar portion of the occipital bone rises with sphenobasilar flexion, the posterior aspect of the petrous portion is raised, tilting its superior ridge anteriorly and laterally. This is also accomplished by raising the jugular process and groove immediately posterior to it. Sutherland³⁶ called this area and the lambdoidal suture a "combined rockshaft and pivot-bearing mechanical arrangement." Elevation of the posterior aspect of the petrous portion and movement of the petrous ridge anteriorly and laterally places the general axis of temporal bone rotation through the petrous portion, which is directed laterally and posteriorly from its apex.

To understand temporal bone movement, it is necessary to have a clear view of what takes place with different sections of the bone as a result of the general axis of rotation through the petrous portion. Magoun²³ relates that Sutherland, in his lectures,

Cranial Osteology

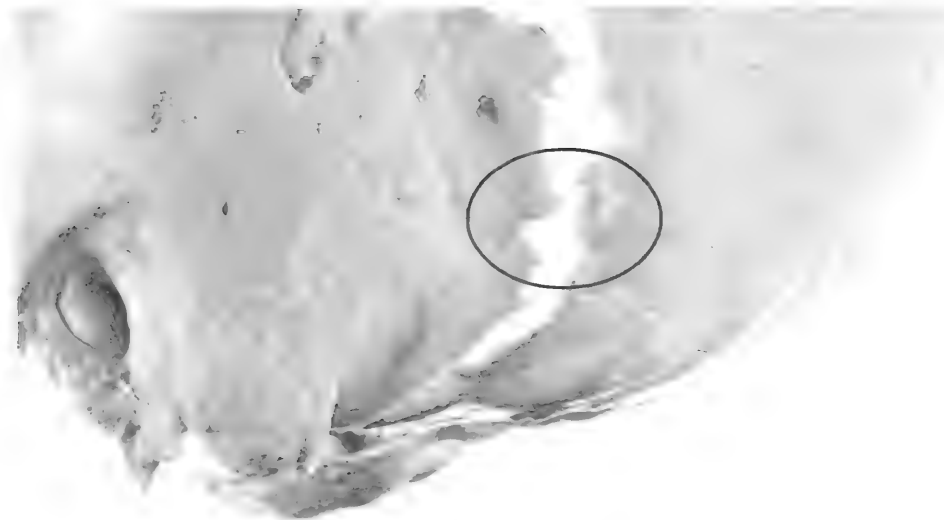


3—49. As the basilar process of the occipital bone rises in inspiration, it lifts the petrous portion of the temporal bone, contributing to the "wobbling wheel" effect of temporal movement.

referred to temporal motion as similar to a "wobbling wheel." This is an apt description. It could be expanded by stating that the axle is not in the center of the wheel. The general axis of temporal bone rotation is toward its inferior and posterior aspects.

To summarize, the general axis of rotation of the temporal bone is off-center and angled anteriorly from the petrous base to its apex. Thus a portion of the bone moves in a particular direction, depending on its relationship with the general axis of rotation. The temporal bone's structures have a three-dimensional motion. Structures above and anterior to the petrous portion move laterally and anteriorly, as does the major portion of the squama. Structures

below — such as the tip of the mastoid process — move posteriorly and medially. The mastoid portion just above and posterior to the root of the petrous portion moves superiorly, anteriorly, and somewhat laterally. Occipital motion appears to move the temporal bone, not only by lifting the petrous portion but by a gear-type action of the occipitomastoid suture above the condylosquamomastoid pivot (CSM). As the occipital bone elevates at this area, it brings the adjacent portion of the temporal bone up by a gear-type action. The occipitomastoid suture below the CSM pivot is serrate, with expanding and tightening motion.

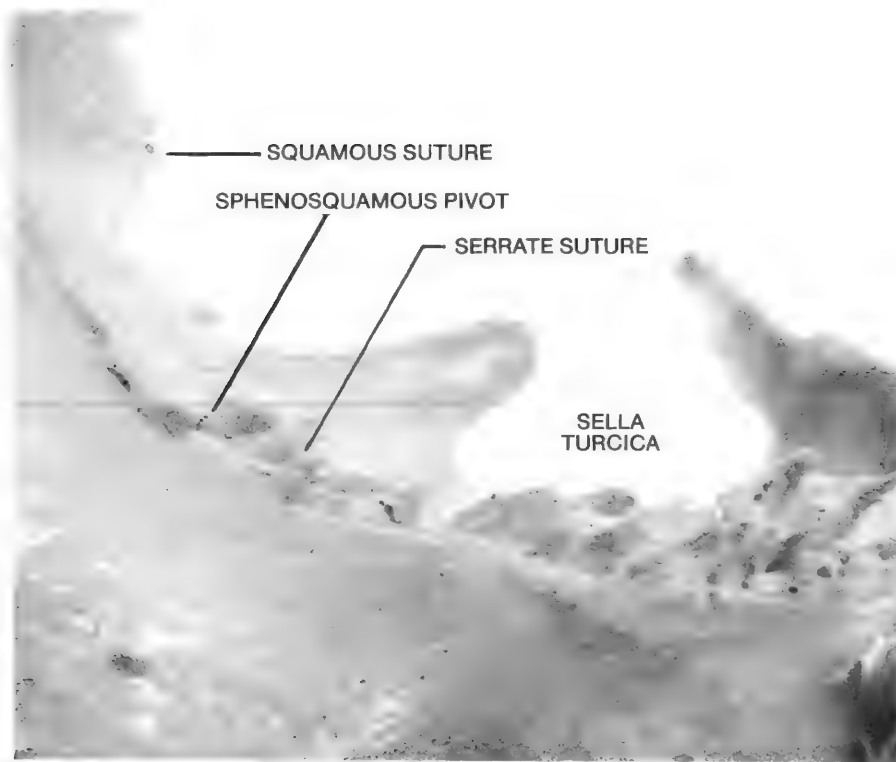


3—51. *Condylosquamosomastoid (CSM) pivot.*

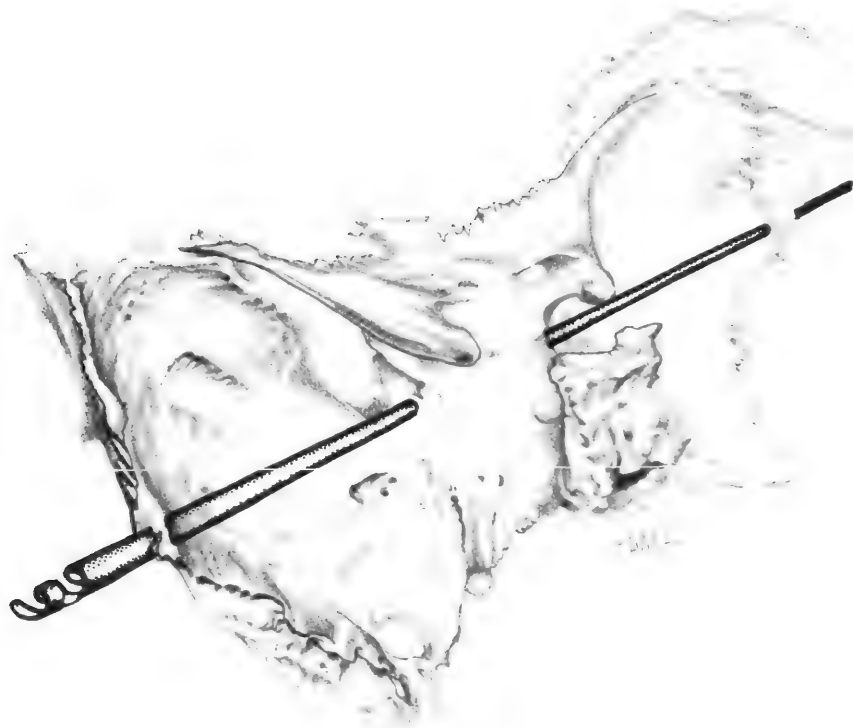
Motion of the temporal bone with the sphenoid varies above and below the spheno-squamous (SS) pivot. This pivot is approximately in the middle of the temporosphenoidal suture. An imaginary line from the right SS pivot to the left gives the general axis of rotation of the sphenoid. The SS pivot is on approximately the same level as the general axis of rotation of the temporal bone, which is through the petrous portion. The SS pivot is necessary for organizing the motion of these two bones with their axes of rotation. Above the SS pivot, the temporosphenoidal suture is squamous, allowing the sliding action necessary for the flaring (external rotation) of the squama of the temporal and the lateral aspect of the greater wing of

the sphenoid. The suture below the SS pivot is serrate, allowing a rocking action between the temporal and sphenoid bones.

The ethmoid completes the basilar portion of the skull formed in cartilage. It is moved by the sphenoid at the sphenoethmoid suture. The posterior aspect of the ethmoid moves inferiorly, while the anterior aspect moves superiorly, the general axis of rotation being inferior to the posterior aspect of the crista galli, slightly above the center line. This motion moves the crista galli posteriorly and superiorly. This is an important point of attachment for the falx cerebri.



3—52. Close-up of the sphenosquamous pivot, which is a limbis suture.



3—53. Sphenosquamous (SS) pivot is the general axis of rotation of the sphenoid bone.

Vault Bones Ossifying From Membrane

The bones of the cranial vault — excluding the basilar portion — ossify directly from membrane, skipping the cartilaginous stage. These are the parietals and the frontal bone, and also the upper squamous portion of the occipital and temporal bones and the lateral aspect of the greater wing of the sphenoid. Before undertaking study of the motion of these bones, the motion of the basilar portion of the cranium mentioned previously should be well understood.

The sagittal suture joins the two parietal bones. This dentate suture has great interdigitation. It acts somewhat as a hinge between the two parietal bones. During sphenobasilar flexion there is slight inferior movement at the sagittal suture and lateral flaring (external rotation) of the lateral border of the parietal, especially that portion in contact with the temporal and sphenoid bones. There is also a slight anterior movement of the parietal bones imparted by the squama and the parietal notches of the temporal bones.

The lambdoidal suture was described by Sutherland³⁶ as having an internal bevel on the occipital bone from the lambda to midway along the suture, and from that point to the junction of the temporal bone having an external bevel. This is generally an accurate description; however, examination of many disarticulated skulls will reveal that some have no bevel along the lambdoidal suture, while others have bevels exactly opposite that which Sutherland described. This is in agreement with the observation in applied kinesiology that there is individuality among skulls; to obtain accurate corrections it is necessary to have a system which considers individually the type of correction necessary so that the physician does not apply inappropriate forces to structures that may not be formed the same as in the majority. Observation of the lambdoidal suture by applied kinesiologists indicates that it is primarily for expansion and tightening, allowing accommodation to motion.

The parietomastoid suture is serrated. This allows the parietal to ride along with the movement of the parietal notch of the temporal bone.

The motion along the squamosal suture and the sphenoparietal is similar. These are broad squamous sutures. There is external rotation of the temporal squama, greater wing of the sphenoid, and the parietal bone, with a major sliding of the suture and a slight anterior motion of the parietal, temporal, and sphenoid bones at this area.

"The frontal bone fits in between the parietals at

its inferior angle. One might view the frontal bone as hung on a hinge at its sagittal junction and having the function of swinging forward at its lower area."³⁶ This description by Sutherland puts into perspective the motion of the frontal bone in its accommodating function relating to other more movable structures. The coronal suture is limbus, having an external bevel on the parietal bone on the central half and an internal bevel on the lateral half. The internal bevel on the lateral half enables the parietal to flare more laterally than the more rigid frontal bone would otherwise allow.

The motion described for the frontal bone is as if the bone maintains the metopic suture throughout life. This motion requires the flexibility of living bone, and appears to be more limited than other cranial bone motion. Rotation appears to be about an axis in the general area of the metopic suture. This gives the metopic suture a hinge-type action; if completely ossified, it has the flexibility of living bone. This analysis is from morphologic study and applied kinesiology evaluation by resisting certain motions as the subject takes various phases of respiration (described on page 87). It appears there is a slight internal movement of the metopic suture area on sphenobasilar flexion, and a lateral anterior movement of the lateral border of the frontal bone. This movement analysis deviates slightly from that described by Magoun.²³ He describes the general axis of rotation as in the center of each orbital plate, projecting through the frontal eminence. This analytical discrepancy does not effectively change the general motion of the frontal bone. In either analysis, the ethmoidal notch widens posteriorly, and the bregma is slightly depressed on sphenobasilar flexion. The glabella moves posteriorly and superiorly with the crista galli.

Another discrepancy is in the terminology used for frontal bone motion. In osteopathic²³ and sacro occipital technique⁶ terminology, external rotation refers to the lateral margin of the frontal bone moving laterally and anteriorly on sphenobasilar flexion. Applied kinesiology refers to external rotation as the metopic suture area moving externally, which takes place on sphenobasilar extension. Internal frontal bone rotation in applied kinesiology is the metopic suture area moving internally, which coincides with external movement of the lateral border and takes place on sphenobasilar flexion. In this text, when internal or external rotation of the frontal bone is mentioned, the portion of the bone being described will be listed to avoid confusion.

Cranial Osteology

Facial Bone Movement

Maxilla: The movement of the maxilla and its association with the vomer and palatine bones are very important, both in cranial motion and in the evaluation and correction of dysfunction.

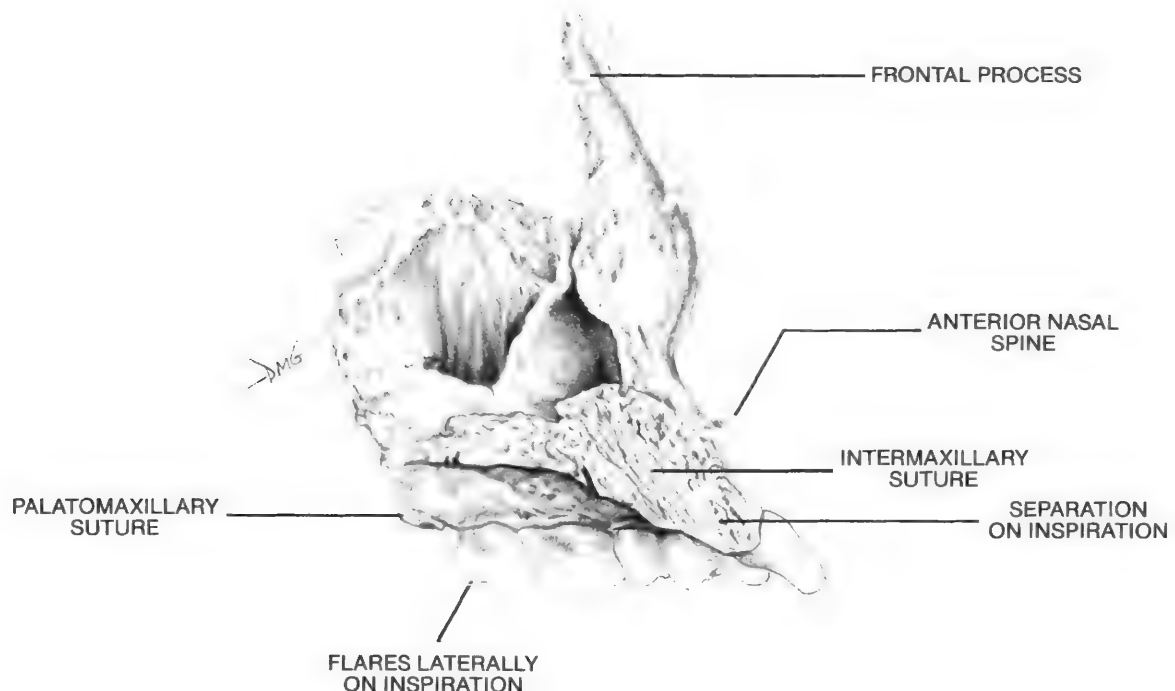
On sphenobasilar flexion, the dental arch flares laterally. This occurs because the intermaxillary suture has a slight hinge-type action in the posterior aspect of the suture and in the superior portion of the anterior suture. The inferior portion of the anterior suture separates slightly. As the teeth flare laterally on sphenobasilar inspiration, the intermaxillary suture drops slightly inferiorly in a similar ratio to the anterior body of the sphenoid. The motion of these two bones is connected by the vomer, which also connects the horizontal plates of the palatine bones with the body of the sphenoid. In this manner the vomer acts as a strut between these bones.

The serrate suture articulation of the maxilla with

the frontal bone stabilizes the maxilla. The serrated nasomaxillary suture sometimes resembles a schindylesis. As the nasal bones move slightly with the ethmoid, there is flexibility of the internasal and frontonasal sutures.

The articulations of the maxillary bone with the ethmoid are plane sutures which, along with the thin, fragile bone making up the ethmoid, provide easy flexibility. Gross distortion of the maxillary bone — usually from birth or other trauma to the infant's skull — can crowd the ethmoid and disturb function.

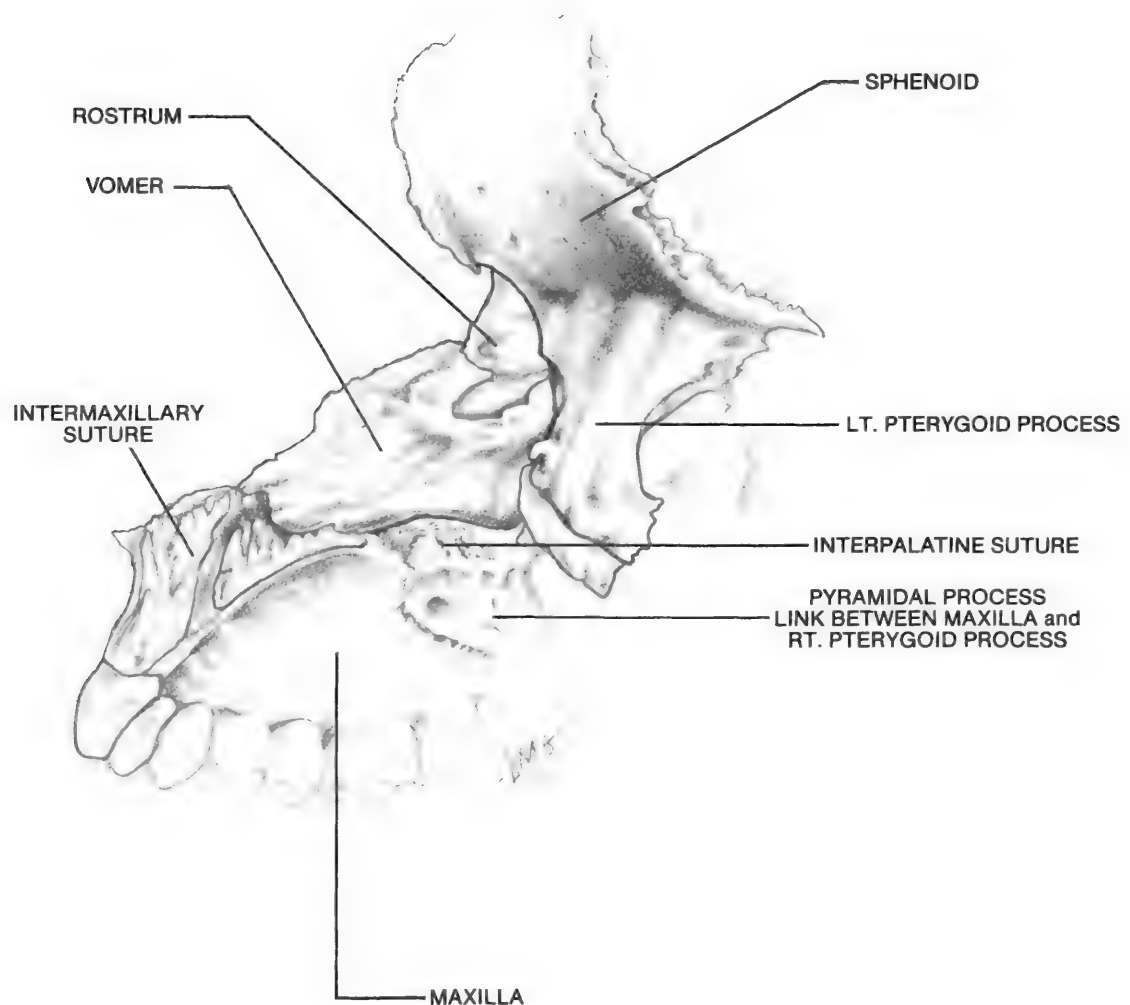
The lacrimal is a very thin, small bone which articulates with the maxilla by the lacrimomaxillary suture. It is a plane suture that articulates the anterior border of the lacrimal with the posterior border of the frontal process, and the inferior border of the lacrimal with the medial aspect of the orbital surface of the maxillary bone.



3—54. *Because of the L-shape of this suture, the hinge action takes place along the upper portion of the suture, while separation takes place at the anterior inferior suture.*

Vomer: The vomer is a very thin, fragile bone which plays an important role in connecting the maxilla and palatine bones with the sphenoid and ethmoid. As the sphenoid and ethmoid move in sphenobasilar flexion, they rotate in opposite directions around their transverse axes. Thus the structures anterior to the general axis of sphenoid rotation move inferiorly, while the structures posterior to the

general axis of ethmoid rotation also move inferiorly. The articulations of the vomer with the rostrum of the sphenoid and with the posterior aspect of the perpendicular plate of the ethmoid, therefore, move the vomer inferiorly to correlate with the inferior movement of the intermaxillary and interpalatine sutures.



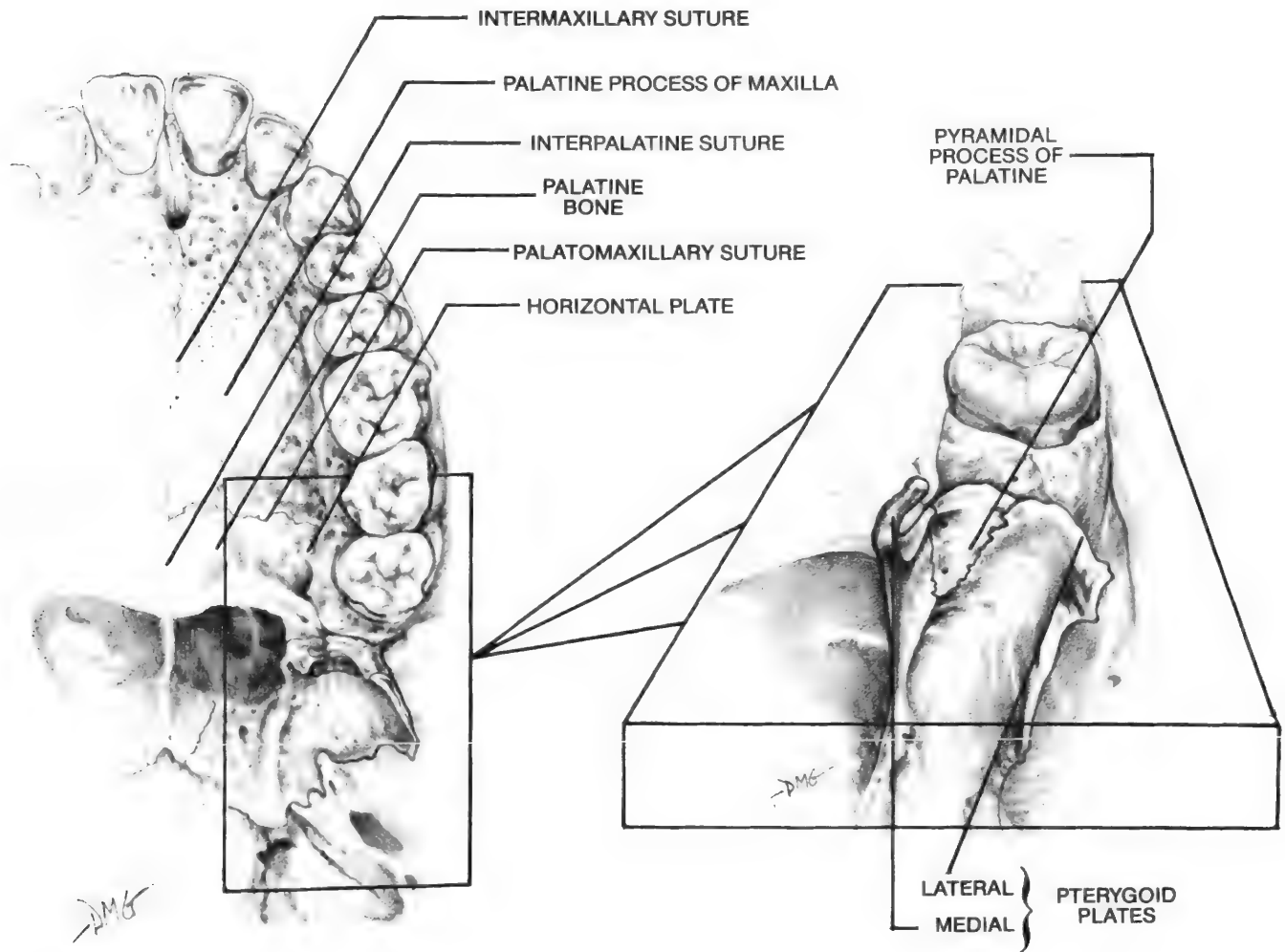
3—55. Connecting mechanism of the sphenoid with the maxilla by way of the vomer. The illustration shows the bones somewhat separated.

Cranial Osteology

Palatine: The palatine bones are important connections between the maxillae and the sphenoid. The interpalatine suture moves inferiorly and slightly posteriorly with the maxillae and vomer. The pyramidal process, because of its location posterior and lateral to the hinge-type motion of the interpalatine suture, moves slightly posteriorly and laterally with the pterygoid process of the sphenoid. Its articulation with the sphenoid at this point is several plane sutures connecting the pyramidal process and the posterior border of the vertical part. The plane sutures provide sliding action to compensate for the disparagement of movement between the pterygoid processes and the palatine bones. The sphenoidal process of the palatine bone articulates with the anterior inferior surface of the sphenoid body by a plane

suture. The orbital process articulates with the anterior portion of the body of the sphenoid.

A serrate suture joins the horizontal plate of the palatine bone with the palatine process of the maxilla. The interpalatine suture is also serrated. Together with the intermaxillary suture and palatomaxillary sutures they form the cruciate suture. The sutures comprising the cruciate suture are often listed as plane sutures in anatomy texts. Although the serration is not as deep as in some serrated sutures, they do have a definite serrated, interdigitated nature which provides strength plus an ability to expand and contract. The pyramidal and maxillary processes of the vertical part join the tuberosity of the maxilla with a strong, serrate suture.



3—56. Pyramidal process connecting mechanisms between maxillary and sphenoid bones.

Zygoma: The zygomatic bone articulates with the maxillary, frontal, and temporal bones with serrate sutures. These all have the ability to expand and contract. The motion is that of adapting to the motion of the cranial and facial bones. The sphenozygomatic suture is limbus, with the external bevel on the sphenoid. The bone moves anteriorly, laterally, and slightly inferiorly on sphenobasilar flexion.

Mandible: The ramus of the mandible moves posteriorly on deep inspiration.^{14, 15} Consideration should be given to the mandible and the temporomandibular articulation regarding skull motion. This will be thoroughly discussed in the section on the TMJ.

Evaluating Skull Motion

Several of the motions just described are the source of considerable controversy among some persons working with the cranial mechanism. A method of evaluating normal cranial motion on an experimental basis appears to be available by using applied kinesiology evaluation. There seems to be no controversy about the superior squama of the temporal bone moving laterally on sphenobasilar flexion (inspiration). This can readily be palpated by those trained in cranial motion palpation, and it has been mechanically measured with mechano-electric transducers by Frymann.⁹

Goodheart^{14, 15} developed a system for evaluating normal skull motion. With the procedure described here, a normal skull can be evaluated for its apparent activity on different phases of respiration. Simply touching an area on a normally functioning skull — such as the squama of the temporal bone — causes no change in a previously strong indicator muscle. Using a subject with no apparent cranial faults, restriction to skull motion can be applied by the examiner. It must be clearly understood that when using this system of evaluation, the individual being tested must be free of any cranial faults as indicated by inspiration or expiration testing and the other methods listed under “Examination and Treatment of Cranial Faults.” If the restriction disturbs normal skull motion, a previously strong indicator muscle will temporarily weaken as observed on manual muscle testing. To evaluate the motion of the temporal squama, place a light pressure (approximately two pounds) on the squamae of both temporal bones while the subject inhales deeply. Release the pressure after the subject reaches maximum inhalation and then test a previously strong indicator muscle; it should weaken immediately and remain weak for varying lengths of time, usually at least fifteen seconds. This is interpreted to mean that restriction of normal motion causes improper afferent stimulation, which is interpreted in the neuronal pools as confusion; thus there is temporary interference with

the body's optimum organization, and a general indicator muscle tests weak on manual muscle testing. The subject's normal respiration without restriction of the skull returns the body to normal function. The same evaluation on the temporal squamae when the subject exhales deeply does not produce a change in muscle strength as evaluated by manual muscle testing.

The body is capable of readily adapting to restriction. If numerous respirations are used in the above demonstration, there will be no observable change when testing an indicator muscle.

Restriction of skull motion in various locations will produce similar results. This appears to be a method by which normal skull motion can be evaluated. If the superior aspect of the occipital squama is restricted from moving posteriorly and slightly inferiorly while the subject inhales deeply, there will be a similar weakening of a previously strong indicator muscle. Motion of the tip of the mastoid process can be evaluated in a similar manner. If the tip of the mastoid process is restricted from moving posteriorly and medially during deep inspiration, a strong indicator muscle will weaken. If the tip of the mastoid process is restricted from moving as before but the subject completely exhales, the indicator muscle will not weaken. This appears to be because the tip of the mastoid process is allowed to move in its normal direction, which is anterior and lateral on exhalation. On the other hand, if the tip of the mastoid process is restricted from moving anteriorly and laterally during a deep inhalation there is no weakening of a previously strong muscle, but there will be if the subject exhales deeply while the restriction is held.

An interesting observation from studying the skull in this manner is that slightly different restriction is necessary in different subjects. Skull individuality is obvious after studying many specimens. It can be observed *in vivo* by the procedure just explained. The variances are very obvious when studying the

Cranial Osteology

sutures of disarticulated skulls. The sutures appear to form in the manner best adapted to motion in that particular skull. Evidence indicates that the cranial primary respiratory motion is active throughout the growth phase of the skull. Since cranial growth takes place at the edge of the bones at the suture,^{2, 24} it seems only reasonable that the growth would be for best movement in that skull, depending on any restrictions dysfunction is causing. In some individuals, a more medial or lateral restriction is necessary in combination with the posterior restriction of the tip of the mastoid process on inhalation to produce

muscle weakness. This reveals the necessity of an approach giving individual consideration to each skull being evaluated for treatment.

The use of this experimental approach to evaluate cranial movement is limited only by the observer's imagination. It can be applied to the pterygoid processes, maxillae, greater wings of the sphenoid, etc.; the only requirement is that the subject have a normally functioning cranial mechanism and associated structures — such as the TMJ and hyoid — and not be neurologically disorganized.

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Chapter 4

Skull Muscles

Muscles Involved With The Stomatognathic System

Structure is almost always correlated with muscle balance in applied kinesiology. This was not the case in early applications of AK to the cranial system. It has been recognized, however, that most cranial faults which return after apparent correction do so because of some type of muscular imbalance. Study of the muscles involved with the stomatognathic system would have little value if it were only a rehash of information available in anatomy texts. Brodie² points this out in his study of the influence of muscles on the stomatognathic system. "The teeth and alveolar processes should be looked upon as passive though responsive victims of a continuous interplay of muscular forces, their positions dictated by the resultants of these forces. No wishful thinking about straight profiles or upright incisors, nor the most clever appliance manipulation, will serve to hold teeth in positions that are contrary to the dictates of their muscular environment." This writer will attempt to correlate what is presented in standard anatomy and physiology texts with the dynamic effects of the muscular system on the total stomatognathic system.

Muscles influencing the stomatognathic system can be divided into six basic groups for consideration: muscles of the cranium and face, masticatory muscles, hyoid muscles, head-on-body muscles, pelvic muscles, and general body muscles. The muscles of the cranium and face are considered here prior to discussion of the types of cranial faults and their examination and correction. The pelvic muscles are considered with the pelvis in Chapter 7, the muscles of mastication in Chapter 9, the head-on-body muscles in Chapters 8 and 10, the muscles of the hyoid in Chapter 13, and finally, general body muscles with Section III on the stomatognathic system's organization in relation to the entire body.

Muscles and Fascia of the Cranium and Face^{1, 7, 9, 11, 12}

Some muscles of the cranium and face can directly create cranial faults or perpetuate them after they have developed. Other muscles presented here are indirectly involved with certain facial expressions or general muscle activity. The physician should use his knowledge of all the cranial and facial muscles during examination. Contraction of certain muscles — as with different facial expressions — puts stress into the cranium in different ways, thus revealing subclinical cranial faults which have not been thoroughly corrected or have gone unrecognized.

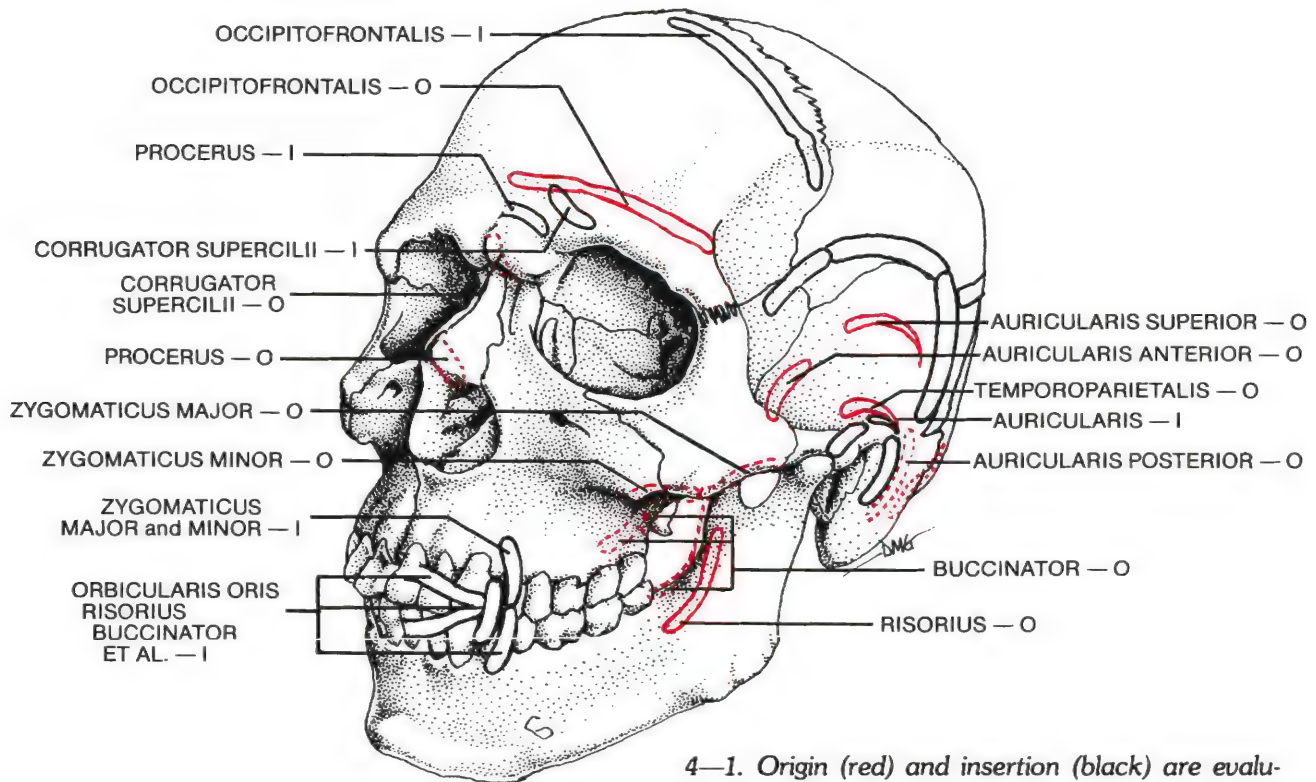
Certain aspects of the fascia are discussed because distal tension placed upon it may either create or perpetuate cranial problems. In a discussion of whiplash injuries, Wright and Brady¹³ state that "... the deep cervical fascia is so continuous with that of the head that the two should be considered as one. The investing layer of the two sides blend together across the midline. It splits to form the sheaths, as that of the sternocleidomastoid muscle in front and the trapezius muscle posteriorly. Also anteriorly it forms the pectoral fascia which in turn blends with the fascial covering of the rectus abdominis." This is in agreement with Rolf's thoughts.¹⁰ She often referred to fascial strain in almost any area of the body causing pressure and pain on a small circular area at the crown of the head. The continuous nature of the fascia throughout the body can place strain between the component parts of the skull from distant structural stress. This agrees with findings in applied kinesiology which show that often structural correction in some other area of the body eliminates cranial faults. It is important to understand the continuous nature of the fascia to appreciate the integration of body function.

Cranial Muscles and Fascia

Subcutaneous and Deep Fascia

The subcutaneous fascia covers the head, investing the facial and cranial muscles. It is a firm, fibrous, fatty layer which adheres to the skin. There is no deep cervical fascia above the superior nuchal and temporal lines or anterior to the masseter muscle, but the deep cervical fascia is so continuous with the superficial fascia of the head that the two should be considered as one.¹³ The subcutaneous fascia of the face and head is continuous with the superficial fascia of the back of the neck. Anteriorly they are directly continuous over the mandible with that of the neck. As noted, the superficial and deep fascia merge toward the epicranium; they are continuous with the deep fascia of the cervical spine, which is continuous with that of the pectoral, deltoid, and back regions. The fascia surrounds the muscles, large blood vessels, and nerves; thus strain can be transmitted throughout the body. There are numerous specific attachments of fascia to centrally placed points on the basilar surface of the skull. The most numerous

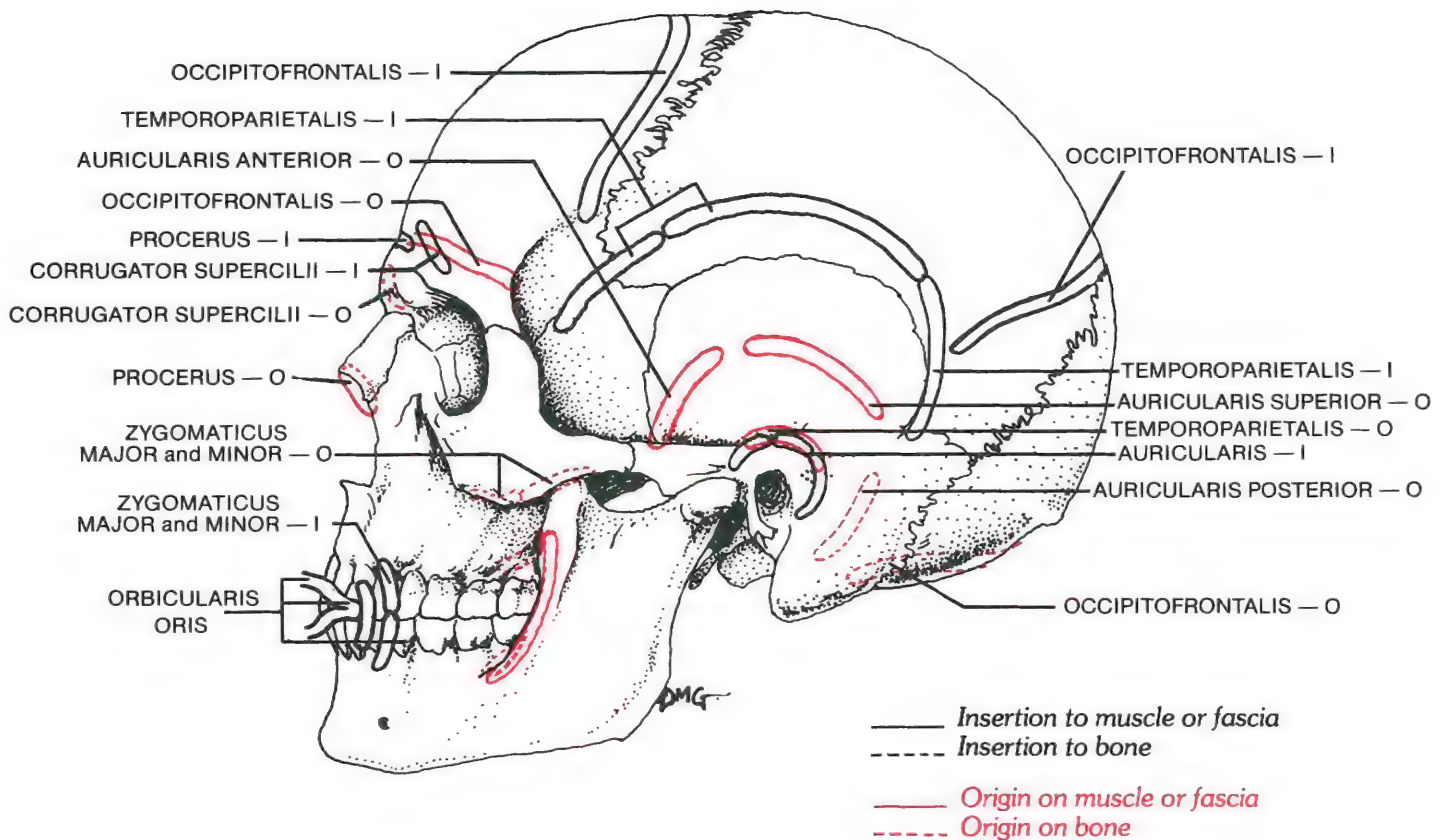
attachments are to the sphenoid, followed by the petrous and squamous portions of the temporal and the basilar surface of the occipital bone.³ These anatomical characteristics reveal a possible explanation of how correction of dysfunction in the modular organization of the body, such as with the PRYT technique, effects changes in the cranial mechanism. It is entirely possible for an individual to have a shoulder involvement as a result of cranial faults, or possibly to have cranial faults as a result of dysfunction in the shoulder. The cause of the involvement may be more remote, such as foot disturbance interfering with proprioceptive function, causing the upper trapezius to be facilitated and inhibited at inappropriate times and thereby creating stress directly in the cranium via its origin. Upper trapezius dysfunction can also cause fascial strain, producing generalized stress to the cranium. These illustrations could go on and on; the important point is to realize that remote structure can involve the cranium through the muscular and fascial systems.



4—1. Origin (red) and insertion (black) are evaluated to determine if a muscle crosses a suture, causing it to more likely affect cranial motion.

—— Insertion to muscle or fascia
 ---- Insertion to bone

—— Origin on muscle or fascia
 ---- Origin on bone



4—2.

OCCIPITOFRONTALIS (Epicranius)

The occipitofrontalis is a broad muscular and tendinous layer which covers the top and sides of the skull from the highest nuchal line of the occipital bone and mastoid portion of the temporal bone to the level of the eyebrows. The muscle portion consists of four parts — two occipital and two frontal — which are connected by the galea aponeurotica.

Occipital Bellies

Origin: From the lateral two-thirds of the highest nuchal line of the occipital bone and from the mastoid portion of the temporal bone.

Insertion: Into the galea aponeurotica.

Action: Draws the scalp posteriorly.

Nerve Supply: Posterior auricular branch of the facial nerve.

Frontal Bellies

Origin: There is no bony origin; rather the fibers blend with the procerus, corrugator supercilii, and orbicularis oculi muscles.

Insertion: Anterior aspect of the galea aponeurotica in front of the coronal suture.

Action: With the anterior aspect of the muscle as the origin, pulls the scalp forward; with the posterior acting as the origin, lifts the eyebrows and skin over the glabella.

Nerve Supply: Temporal branches of the facial nerve.

Skull Muscles

Galea Aponeurotica (Epicranial Aponeurosis)

The galea aponeurotica completes the occipitofrontalis by forming a cover for the upper part of the cranium with the frontal and occipital bellies. At its posterior, it attaches to the external occipital protuberance and the highest nuchal line between the occipital bellies. Anteriorly, it sends a prolongation between the frontal bellies. The auricularis anterior and superior muscles are attached to the lateral borders of the galea aponeurotica, as are the temporoparietalis muscles.

The galea aponeurotica is firmly attached to the skin by way of the superficial fascia. It is separated from the pericranium by the fascial cleft, allowing movement of the integument over the skull for a considerable distance. The combination of the movement and the padding afforded by the subcutaneous tissue and galea aponeurotica provides considerable protection for the skull. Without this protection, as little as forty pounds per square inch can fracture the skull; in the intact cadaver it requires 425-900 pounds per square inch to fracture the underlying skull.⁸

Test: The patient raises his eyebrows as far as possible. The physician should observe for symmetry of the forehead wrinkles to determine the balance of the muscles bilaterally.

Discussion: Hypertonicity of the occipital or frontal bellies can cause jamming of the coronal or lambdoidal sutures. General tightening of the cranial fascia can influence other areas, such as the temporal bone. Lack of muscle activity may contribute to separation of the sutures.



4—3. Imbalance of relaxed occipitofrontalis.



4—4. Near normal contraction of occipitofrontalis; there is slightly greater contraction on the right.

TEMPOROPARIETALIS

Origin: From the temporal fascia above and anterior to the ear. It spreads like a fan in three parts.

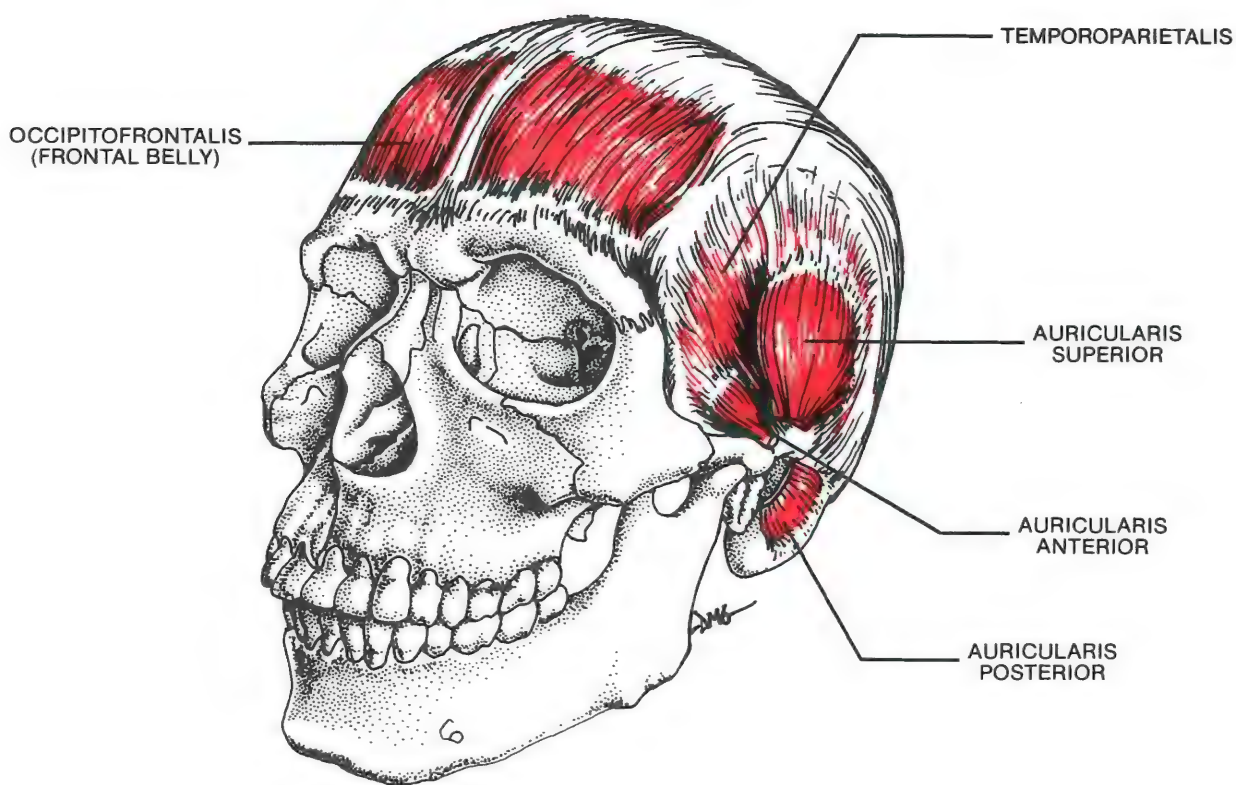
Insertion: Lateral border of the galea aponeurotica in three sections: (1) anterior — temporal part; (2) medial — triangular part; and (3) superior — parietal part.

Action: Tightens the scalp; combined with the occipitofrontalis, draws back the skin of the temples.

Nerve Supply: Temporal branches of facial nerve.

Test: Most subjects cannot adequately contract the sections of this muscle for evaluation. The sections can be palpated for tension, and therapy localization can be used to help locate dysfunctioning proprioceptors.

Discussion: In all probability, treatment to influence this muscle will be combined with the auricularis muscles listed next. Attention should be given when there is a recurrent jamming of the sagittal or squamosal sutures.



4—5.

Extrinsic Muscles of the Ear

AURICULARIS ANTERIOR

Origin: Temporal fascia.

Insertion: Projection on the front of the helix.

Action: Draws the ear forward.

Nerve Supply: Temporal branches of the facial nerve.

AURICULARIS SUPERIOR

Origin: Fascia of temporal region.

Insertion: Upper part of the medial surface of the auricle.

Action: Draws ear upward and tightens galea aponeurotica.

Nerve Supply: Temporal branches of the facial nerve.

AURICULARIS POSTERIOR

Origin: Mastoid portion of the temporal bone.

Insertion: Cranial surface of the auricular concha.

Action: Draws ear posteriorly.

Nerve Supply: Posterior auricular branch of the facial nerve.

Test: Testing these muscles by asking the subject to move an ear is problematic since a high percentage of people cannot voluntarily move their ears in various directions. An improved method of evaluating these muscles was devised by Goodheart.^{5, 6} The examiner pulls the ear in various directions to stretch either the anterior, superior, or posterior auricularis muscle. A previously strong indicator muscle is tested for weakening, which indicates the muscle is hypertonic. A correlation with cranial function can be observed by having the patient take a phase of respiration to determine if the indicator muscle immediately regains its strength. Whatever phase of respiration abolishes the challenge indicates the type of cranial fault involved.

Discussion: These are very small, delicate, and indistinct muscles. They may make some contribution to sagittal or squamosal suture faults, but the amount of contribution is questionable.

Muscles of the Eyelids

ORBICULARIS OCULI

The orbicularis oculi is the sphincter muscle of the eyelids. It is a broad, flat, elliptical muscle surrounding the circumference of the orbit and consisting of three parts — the orbital, palpebral, and lacrimal.

Orbital Part

Origin: From the medial orbital margin which is made up of the frontal and maxillary bones, and from the medial palpebral ligament. The medial palpebral ligament is about 4 mm in length and 2 mm wide; it attaches to the frontal process of the maxilla in front of the nasolacrimal groove.

Insertion: The fibers arch around the upper lid, making an ellipse by returning around the lower lid to the medial palpebral ligament. Some of the fibers along the upper lid blend with the frontal belly of the occipitofrontalis, the corrugator supercilii, and into the skin and subcutaneous tissue of the eyebrow. These fibers make up the depressor supercilii.

Action: The strongest portion is used to tightly close the lids or to wink with one eye.

Nerve Supply: Temporal and zygomatic branches of the facial nerve.

Test: The eyelid is forcefully closed. This should be strong enough to put pressure on the eyeball, as well as cause wrinkles at the outer edge of the eye. This test is used in conjunction with a previously strong indicator muscle to challenge some of the bones making up the eye socket.

Palpebral Part

Origin: From a similar origin, the fibers progress across the eyelid straighter than the orbital part.

Insertion: Into the lateral palpebral raphe.

Action: Closes the lid gently as in blinking or in sleep.

Nerve Supply: Temporal and zygomatic branches of the facial nerve.

Test: Observe the patient's ability to gently close his eyelid, as in blinking.

Lacrimal Part

Origin: Lacrimal fascia and upper part of the lacrimal bone. Passes laterally behind the lacrimal sac, dividing into upper and lower slips.

Insertion: Into the medial portion of the upper and lower eyelids.



4—6. Orbicularis oculi test. Note imbalanced contraction of the procerus and corrugator supercilii muscles. Also, there is imbalance of the shoulders and neck muscles.

Action: Active in the propulsion of tears.

Nerve Supply: Temporal and zygomatic branches of the facial nerve.

Discussion: Action of all parts of the orbicularis oculi tends to tighten the sutures of the orbital rim. This can be used in evaluation to determine if cranial faults are present in this area.

Tightening of the orbicularis oculi may also put pressure on the orbital cavity, thus challenging the sutures and bones deep within the structure. Change of muscular strength on its tight contraction indicates further evaluation of the bones making up the orbit is necessary.

The medial palpebral ligament is tightened through the action of the orbicularis oculi each time the eyelids are closed. This draws the lacrimal sac laterally and forward, creating a vacuum so that tears are sucked along the lacrimal canals; the lacrimal sac is compressed to deliver tears for the eye. When there is a lack of tears, cranial faults should be considered. The orbicularis oculi receives its motor supply from the facial nerve (cranial VII), which also supplies parasympathetic secretomotor fibers to the lacrimal gland. On a functional basis, lack of tearing is generally heard as a complaint from those wearing contact lenses. Correction of cranial faults often improves this condition.

CORRUGATOR SUPERCILII

Origin: Medial part of the supraorbital margin.

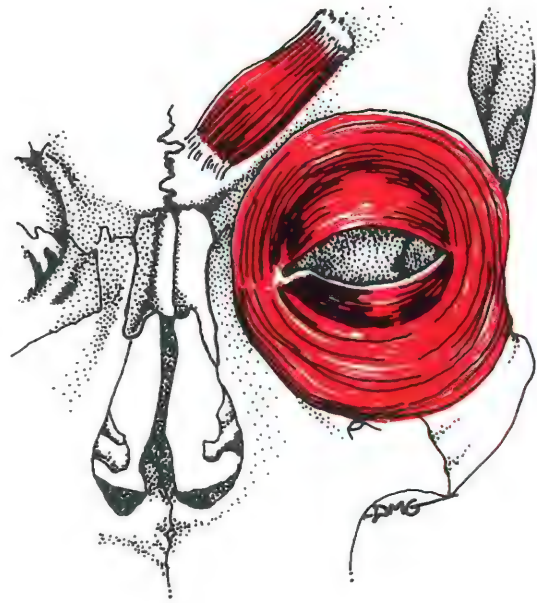
Insertion: Skin of the medial half of the eyebrow.

Action: Draws eyebrow downward and medially. Bilateral action produces vertical wrinkles at glabella; the principle muscle in frowning.

Nerve Supply: Temporal branch of the facial nerve.

Test: Observe the patient's ability to bring the eyebrows downward and medially, as in frowning. Observe for the symmetry of the vertical wrinkles which are produced.

Discussion: Imbalanced activity, in comparing one side with the other, causes the vertical wrinkles of the glabella to be off-center. The corrugator supercilii, in conjunction with the procerus muscle, may be involved in some conditions of nasal congestion.^{5,6}



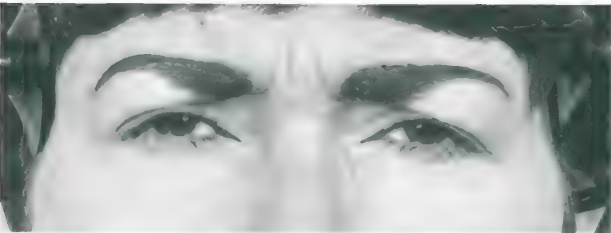
4—10. *Corrugator supercilii and orbicularis oculi.*



4—7. *Relaxed position of corrugator supercilii.*



4—8. *Contraction of corrugator supercilii. Note movement of eyebrows. There is absence of lines in the skin of a young person.*



4—9. *Deep lines and some imbalance.*

LEVATOR PALPEBRAE SUPERIORIS

Origin: Inferior surface of the lesser wing of the sphenoid.

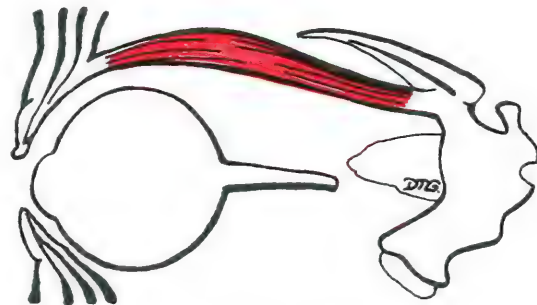
Insertion: To the lamellae, both superficial and deep.

Superficial lamella: superior tarsus, orbicularis oculi, and to the skin of the upper eyelid.

Deep lamella: superior tarsus.

Action: Antagonistic to orbicularis oculi to hold the upper eyelid open.

Nerve Supply: Sympathetic from the superior division of the oculomotor (III).



4—11. *Levator palpebrae superioris.*

Muscles of the Nose

PROCERUS

Origin: Fascia of upper part of lateral nasal cartilage and lower parts of nasal bone.

Insertion: Into the skin over the lower part of the forehead between the eyebrows. Fibers decussate with those of the frontal belly of the occipitofrontalis.

Action: Draws down the medial angle of the eyebrows and produces transverse wrinkles over the glabella. Pulls the skin of the nose superiorly.

Nerve Supply: Buccal branches of the facial nerve.

Test: Observe the patient's ability to draw the skin of the nose up and the medial angle of the eyebrows down. This should produce symmetrical transverse wrinkles across the nose.

Discussion: Hypertonicity of this muscle with the corrugator supercilii is often associated with nasal congestion. Goodheart has also clinically observed an association of this muscle with lymphatic congestion of the small intestine, as indicated by quadriceps or abdominal muscle weakness.^{5,6}



4—14. Relaxed position.



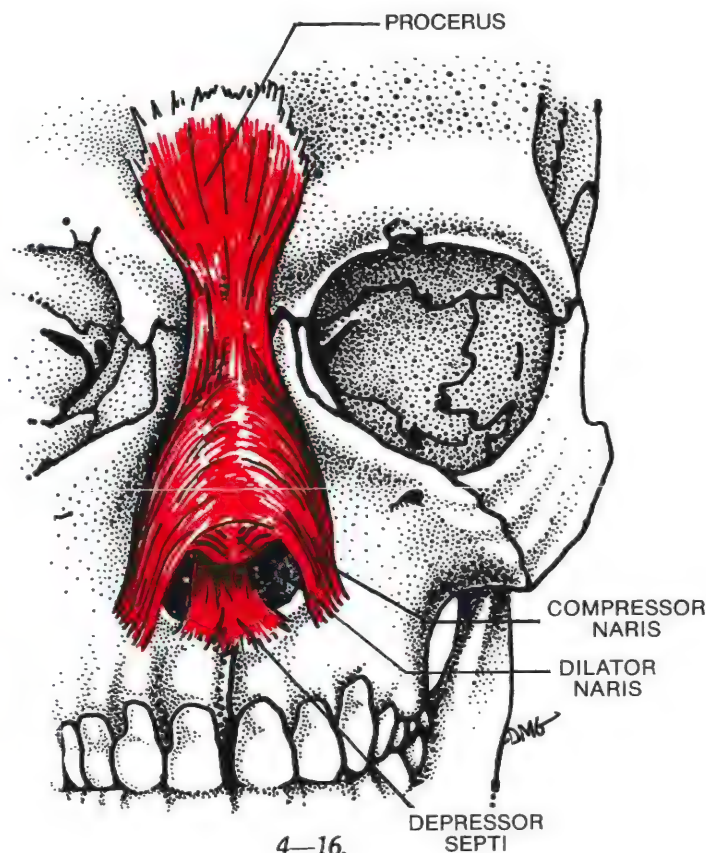
4—15. Procerus muscle contraction with some contraction of the corrugator supercilii.



4—12. Relaxed face showing body language of chronic procerus hyperactivity or hypertonicity.



4—13. Same subject as above with only facial expression as mechanism of contraction.



4—16.

COMPRESSOR NARIS
(Nasalis, Transverse Part)

Origin: From the maxilla above and lateral to the incisive fossa.

Insertion: Into an aponeurosis continuous with the muscle of the opposite side.

Action: Compresses the nasal aperture.

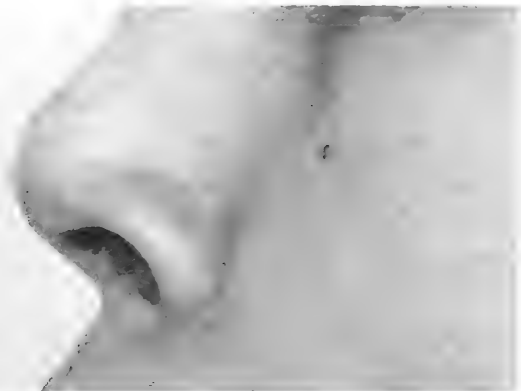
Nerve Supply: Buccal branches of the facial nerve.

Test: Observe the patient's ability to draw the point of the nose down, compressing the nostrils. The action should be symmetrical.

Discussion: Strong contraction may tighten the anterior intermaxillary suture. If a previously strong indicator muscle weakens on contraction of this muscle, consider the possibility of a jammed intermaxillary suture. The muscle is usually not well developed and is rarely involved with cranial faults.



4—17. Relaxed position.



4—18. Compressor naris contraction. Many cannot contract this muscle.

DEPRESSOR SEPTI

Origin: From the maxilla above the central incisor tooth.

Insertion: Mobile portion of the nasal septum.

Action: Draws ala of the nose downward.

Nerve Supply: Buccal branches of the facial nerve.

Test: Muscle is tested with the compressor naris.

DILATOR NARIS
(Nasalis, Alar Part)

Origin: Margin of the nasal notch of the maxilla below and medial to the transverse part.

Insertion: Skin near the margin of the nostril.

Action: Draws ala downward and laterally to assist in widening the anterior aperture. Combats the tendency of atmospheric pressure to close the nostril in deep inspiration.

Nerve Supply: Buccal branches of the facial nerve.

Test: Observe the patient's ability to flare the nostrils, increasing the diameter.

Discussion: The contraction does not appear to correlate with any particular cranial fault. Test can be used to help evaluate the facial nerve.



4—19. Dilator naris prior and after contraction. Some patients have difficulty contracting this muscle.

Muscles of the Mouth

The mouth has a complex arrangement of muscles, giving it the range of motion and flexibility needed for speaking and in mastication. Some of the muscles correlate directly with cranial faults, while others change the tension on the fascia investing the face and head.

LEVATOR LABII SUPERIORIS

Origin: From the maxilla and zygomatic bones at the lower margin of the orbital opening.

Insertion: Into the muscle of the upper lip between the levator anguli oris and the levator labii superioris alaeque nasi.

Action: Raises and everts the upper lip.

Nerve Supply: Buccal branches of the facial nerve.

Test: Test is with levator labii superioris alaeque nasi.

Discussion: May be involved with the zygomatico-maxillary suture.

LEVATOR LABII SUPERIORIS ALAEQUE NASI

Origin: Upper part of the frontal process of the maxilla.



4—20. Levator labii superioris alaeque nasi and levator labii superioris tested bilaterally. Note lack of increase of nasolabial fold and lip elevation on the left.

Insertion:

Medial slip: Greater alar cartilage and skin of the ala of the nose.

Lateral slip: Into the levator labii superioris and orbicularis oris.

Action:

Medial slip: Dilates the nostril.

Lateral slip: Aids in raising and everting the upper lip.

Nerve Supply: Buccal branches of the facial nerve.

Test: The levator labii superioris alaeque nasi is tested bilaterally by observing the patient's ability to raise the upper lip as if to reveal the upper gingiva.

LEVATOR ANGULI ORIS

Origin: From the canine fossa of the maxillary bone.

Insertion: Angle of the mouth intermingling with fibers of the zygomaticus major, depressor anguli oris, and orbicularis oris.

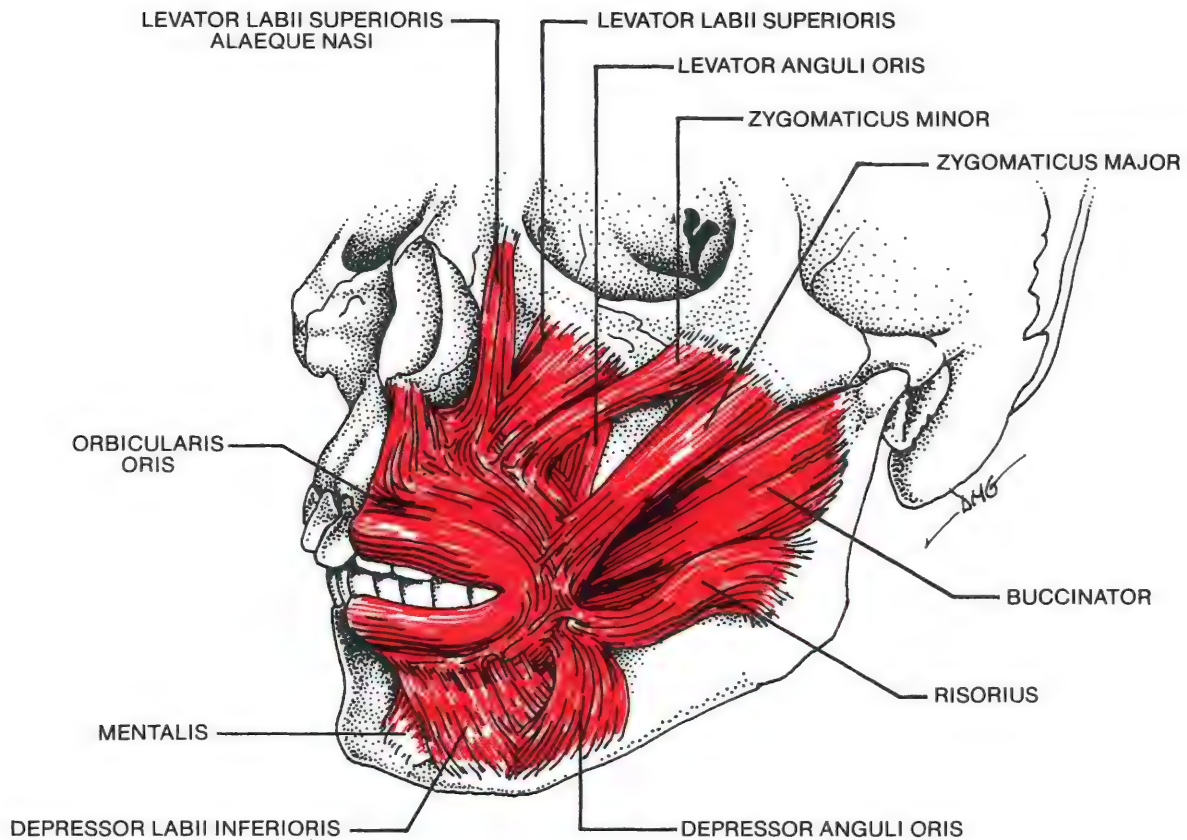


4—21. Unilateral and bilateral levator anguli oris contraction. This subject represents poor ability to isolate the muscle action.

Action: Raises the angle of the mouth and helps produce the nasolabial fold.

Nerve Supply: Buccal branches of the facial nerve.

Test: Observe the patient's ability to lift the angle of the mouth straight upward. This is the action that would open the lips over the canine tooth. The test is



4—22.

best done unilaterally to help eliminate the levator labii superioris and the levator labii superioris alaeque nasi. Compare the deepening of the nasolabial folds from muscle contraction.

Discussion: The muscle does not cross a suture. It is tested most frequently to evaluate the facial nerve.

ZYGOMATICUS MINOR

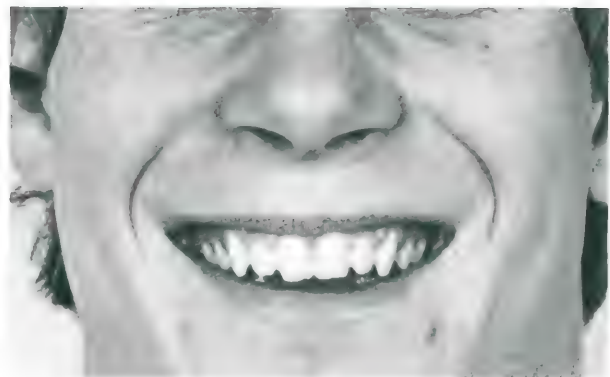
Origin: Lateral surface of the zygomatic bone immediately behind the zygomaticomaxillary suture.

Insertion: Muscle substance of the upper lip between levator labii superioris and the zygomaticus major.

Action: Elevates upper lip and assists in producing the nasolabial fold.

Nerve Supply: Buccal branch of the facial nerve.

Test: Test is primarily with the zygomaticus major, described next.



4—23. Zygomaticus major and minor contraction. The face is at an exact right angle with the camera. Note less contraction on the left.

Skull Muscles

ZYGOMATICUS MAJOR

Origin: Zygomatic bone anterior to the temporozygomatic suture.

Insertion: Angle of the mouth blending with the fibers of the levator and depressor anguli oris and orbicularis oris.

Action: Draws the angle of the mouth upward and laterally as in laughing.

Nerve Supply: Buccal branch of the facial nerve.

Test: Observe the patient's ability to draw the angle of the mouth posteriorly and superiorly as in laughing. The zygomaticus minor has a more medial insertion, while the zygomaticus major inserts directly into the angle of the mouth. Compare for symmetry of muscle action.

Discussion: Both the zygomaticus major and minor cross the zygomaticomaxillary suture. Their hypertonicity can cause jamming of the suture, while their weakness may cause it to spread. Both muscles deepen the nasolabial fold.



4—24. Deepened nasolabial fold in a relaxed individual. It is deeper on the left, and there is some left eye protrusion.

RISORIUS

Origin: Fascia over the masseter.

Insertion: Skin at the angle of the mouth.

Action: Retracts the angle of the mouth.

Nerve Supply: Buccal branch of the facial nerve.

Test: Observe the patient's ability to draw the angle of the mouth laterally and posteriorly, with minimal elevation.

Discussion: Weakness or hypertonicity may possibly influence the maxilla or mandibular activity.



4—25. Good contraction of the risorius muscles. Note flare of nostrils from dilator nares contraction; there is greater contraction on the right.

DEPRESSOR LABII INFERIORIS

Origin: Oblique line of the mandible between the symphysis menti and mental foramen.

Insertion: Skin and orbicularis oris of the lower lip; some blending with the muscle of the opposite side.

Action: Draws lower lip downward and slightly laterally.

Nerve Supply: Mandibular and buccal branches of the facial nerve.

Test: Observe patient's ability to draw lower lip inferiorly, as if to uncover the lower anterior teeth. This action is aided by the anterior fibers of the platysma.



4—26. Less contraction of the depressor labii inferioris on the left. There is also contraction of the mentalis.

DEPRESSOR ANGULI ORIS

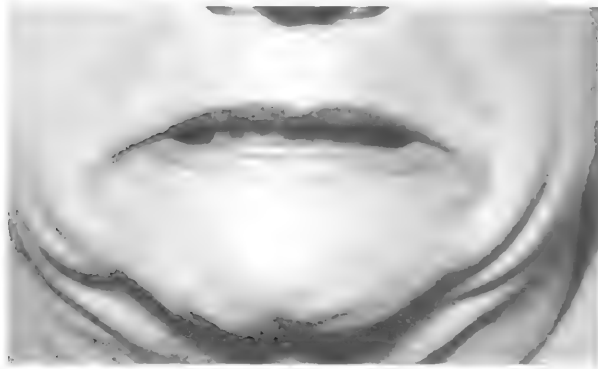
Origin: Oblique line of the mandible, lateral to the mental foramen.

Insertion: Angle of the mouth, blending with the other muscles.

Action: Draws the angle of the mouth downward and laterally; it is the antagonist of the levator anguli oris.

Nerve Supply: Mandibular branches of the facial nerve.

Test: Observe the patient's ability to draw the angles of the mouth downward. In observing this, care must be taken that the patient is not elevating the central portion of the lower lip by contraction of the mentalis, which raises and protrudes the lower lip.



4—27. *Depressor anguli oris contraction with platysma synergism.*

MENTALIS

Origin: Incisive fossa of the mandible.

Insertion: Into the integument of the chin.

Action: Raises and protrudes the lower lip.

Nerve Supply: Mandibular branch of the facial nerve.

Test: Observe the patient's ability to raise and protrude the lower lip.

BUCCINATOR

Origin: There are three areas of origin — from the maxilla, pterygomandibular raphe, and the mandible.

Maxilla: From the buccal surface of the alveolar processes of the three molar teeth.

Pterygomandibular raphe: This is a tendinous inscription between the buccinator and constrictor pharyngis superior muscles from which the middle portion of the muscle originates. It is attached superiorly to the pterygoid hamulus and inferiorly to

the posterior end of the mylohyoid line of the mandible.

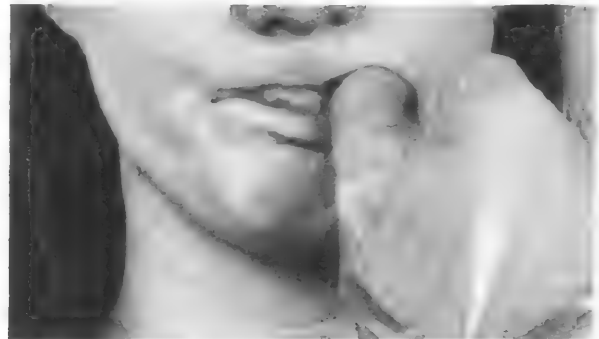
Mandibular: The lateral alveolar processes inferior to the three molar teeth.

Insertion: Blends into the deeper layer of muscle fibers of the lips; splits in the middle to join with the orbicularis oris of the upper and lower lips.

Action: During mastication the buccinator acts to hold food between the teeth. It is a major component of the outer muscular envelope of the dental arches. Compresses cheeks to forcefully blow air, such as in trumpet playing.

Nerve Supply: Buccal branches of the facial nerve.

Test: Observe the patient's ability to contract the buccinator, placing external pressure against the lateral dental arches. Palpate the muscle for contraction. Another evaluation is to have the patient purse the lips and apply maximum air pressure to them. Palpate the buccinator bilaterally for contraction. An even better method is for the physician to place his index finger between the teeth and the cheek, and have the patient muscularly contract against the examining finger. Palpate for contraction of the buccinator.



4—28. *Testing buccinator muscle contraction.*



4—29. *Mentalis muscle contraction.*

Skull Muscles

Discussion: The buccinator appears to be involved on a reactive muscle basis with the masseter muscle in some cases of temporomandibular joint dysfunction. This same reactive muscle basis may be present with some of the smaller muscles of the lips, such as the depressor anguli oris, mentalis, zygomaticus, orbicularis oris, etc. The buccinator muscle is an important part of the outer muscular envelope of the dental arches. Not only does it supply lateral support, but it is also a major base from which the orbicularis oris functions.

ORBICULARIS ORIS

Origin and Insertion: The orbicularis oris is a complex sphincter muscle with fiber contribution from many muscles. The deeper layer receives many fibers from the buccinator, with some of the fibers at the area dividing and decussating so that upper fibers go to the lower lip and lower fibers go to the upper lip. A superficial layer to this area is formed by the levator and depressor anguli oris, which cross each other at the angle of the mouth. This causes the fibers from the levator to pass to the lower lip, while those from the depressor go to the upper lip. Fibers are also derived from the levator labii superioris and levator labii superioris alaeque nasi, the zygomaticus major and minor, and the depressor labii inferioris.

Action: Closes the lips with compression and contraction. With aid of the associated muscles protrudes the lips and contributes considerably to facial expression. Both the superior and inferior aspects are major contributions to the outer muscular envelope of the dental arches.

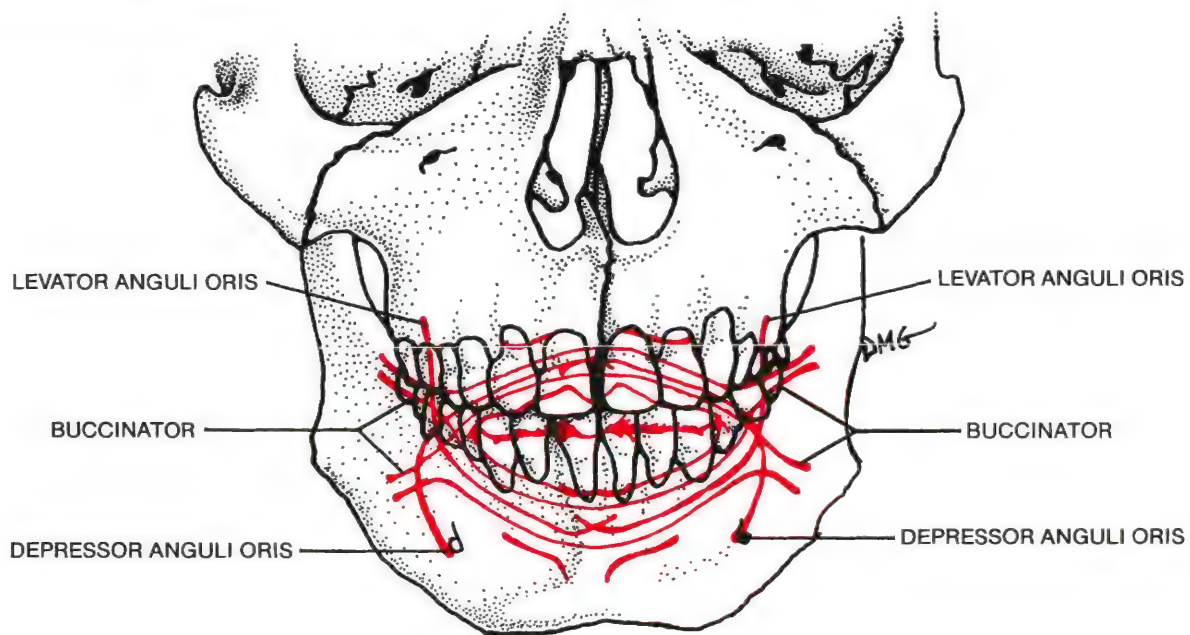
Nerve Supply: Buccal branches of the facial nerve.

Test: Observe the patient's ability to tighten the lips and protrude them. A test of actual strength is described by Garliner.⁴ A button with a string attached is placed posterior to the lips so that the individual holds the button behind the lips with contraction of the orbicularis oris. A spring-force scale is attached to the string to read the pounds of force necessary to pull the button from behind the lips.



4—31. Orbicularis oris contraction. Note slight shift to the left.

Discussion: The strength of the orbicularis oris is important in orofacial balance, as described by Garliner. His research indicates that from three to five pounds of resistive lip strength are normal for most age groups. Strength above or below is considered abnormal.



4—30.



4—32.



4—33.

Figure 4—32 appears mostly normal for an orbicularis oris test. There is a small amount of left eye closing. It would probably be overlooked in most examinations. Figure 4—33 is a test of risorius action. Note contraction of the orbicularis oris and other muscle contraction which was spontaneous and could not be controlled when the risorius was contracted. This involuntary contraction of another muscle is called synkinesis. This patient did not respond to a therapeutic trial of cranial fault correction and was referred for neurosurgery, which was successful (see page 124).

Other Muscles Influencing the Cranium

MUSCLES OF MASTICATION

The muscles of mastication are very important in cranial function. They will be mentioned just briefly here because they are covered thoroughly in the temporomandibular joint section of this text. The muscles directly associated with mastication are generally considered to be the masseter, temporalis, and the internal and external pterygoid muscles. They are very powerful muscles, and because of their origins, insertions, and actions they play an important role in applying pressures to the skull. An imbalance of muscle action or occlusion can create or perpetuate cranial faults.

HYOID MUSCLES

The hyoid muscles are also important to the cranium, and by applied kinesiology examination they appear to have important postural roles which are less recognized in standard physiology. These

muscles are the digastric, stylohyoid, mylohyoid, geniohyoid, sternohyoid, thyrohyoid, omohyoid, and hyoglossus. They will be discussed in the hyoid section of this text.

POSTURAL MUSCLES

Many of the major postural muscles — such as the sternocleidomastoid, upper trapezius, semispinalis capitis, splenius capitis, longus capitis, longissimus capitis, etc. — have a direct bearing on cranial function. There is also an indirect correlation with cranial function by postural muscles remote from the skull, such as those that balance the pelvis, general spinal muscles, etc. (Most of these muscles are covered in Volume I of this series of applied kinesiology texts. They will be correlated to cranial function in the latter portion of this text, where the organization of the stomatognathic system in relation to the entire body is described.)

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Chapter 5

Cranial Nerves

Cranial Nerve Introduction

Apparent cranial nerve involvement is often observed in dysfunction of the cranial primary respiratory mechanism. Some conditions can be traced directly to cranial nerve dysfunction by the usual neurologic testing procedures. More often the apparent cranial nerve dysfunction is observed clinically by the dysfunction of organs, glands, or muscles supplied by the nerve. Direct or indirect testing of the supplied structure before and after cranial correction indicates the nerve's probable involvement.

The physician working with this system must be thoroughly aware of the various aspects that can influence the cranial nerves, and know how to do a thorough evaluation of the system to determine probable causes of various types of dysfunction. This differential diagnosis is particularly important since many patients seek treatment because of trauma which could have caused a cranial fault, or structural damage which would require other forms of treatment. Examination should consider the possibility of fracture, subdural hemorrhage, concussion, damage to a nerve at an encroaching area (such as its foramen), infection, as well as many other possibilities. The physician studying this text should already have diagnostic acumen to evaluate trauma and symptoms relating to the head. In addition, neoplasms, metabolic and degenerative diseases, etc., should be considered and differentiated in the evaluation to rule out these possibilities. In addition to the usual neurologic and other methods of

evaluating the cranial nerves and head, applied kinesiology techniques supply the physician with information which helps determine the extent of involvement and whether it is a functional or pathological problem.

Many aspects of nerve entrapment are considered by various schools of thought. Some approaches consider entrapment when there is only subtle irritation on the nerve causing functional disorders, while others limit the term only to severe damage of the nerve. Entrapment neuropathy is referred to by Kopell and Thompson³² as " . . . a region of localized injury and inflammation in a peripheral nerve that is caused by mechanical irritation from some impinging anatomical neighbor. It may occur at the point at which a nerve goes through a fiber or osseofibrous tunnel, or where the nerve changes its course over a fibrous or a muscular band. Although force may have been applied directly to the region, in many cases there is no discernible relationship of the condition to external trauma." Entrapment neuropathies are often considered as conditions requiring surgery or major structural change in order to provide "release" of the nerve involved.

One of the most commonly recognized entrapment neuropathies is the carpal tunnel syndrome. Generally recognized treatment for this condition, in the absence of neoplasms, is removing the mechanical source of continued irritation. This could be occupational, the use of anti-inflammatory agents, or rest and immobilization. If these conservative methods

Cranial Nerves

do not work, then decompression by surgical intervention is recommended.^{32, 70} This standard procedure for a common nerve entrapment could be radically changed if more physicians were aware of the applied kinesiology approach to this condition. In the absence of neoplasms or structural change — such as a Colles fracture — this condition responds dramatically without anti-inflammatory drugs, surgery, or even rest in most cases. The treatment does require accurate evaluation with challenge and therapy localization. Also, the muscles involved which may be weak and contributing factors — such as the pronator quadratus — must be evaluated and corrected. Usually exceptional response takes place even when there is significant atrophy of the opponens pollicis muscle, generally an indication for surgical decompression. (This condition is thoroughly discussed in Volume IV.)

In some ways the peripheral nerve entrapment recognized in applied kinesiology from cranial primary respiratory mechanism dysfunction parallels that of the carpal tunnel syndrome. When considering the skull it is much more difficult to recognize and evaluate with both clinical and laboratory testing procedures, such as nerve conduction studies. It appears that many of the symptomatic patterns which develop as a result of cranial faults are from subtle impingement upon nerves, creating a type of nerve entrapment. Although subtle mechanical irritation has been shown to change function of the sciatic nerve in mice,⁶⁹ there have been no similar reports of possible irritation from cranial primary respiratory dysfunction on the cranial nerves. This is due to the complexity and inaccessibility of the mechanism, similar to the complexities that have delayed the analysis of the somatoautonomic reflexes.^{59, 60}

In cranial dysfunction the subtle type of nerve entrapment referred to here is such that it often appears to cause significant dysfunction in cranial nerve activity, both efferent and afferent; however,

the problem is reversible, often immediately, with the proper therapeutic approach. The physician who understands standard methods of evaluating cranial nerve and brain function for conditions such as neoplasms, subdural hemorrhage, fractures, etc., and also understands the applied kinesiology approach to functional disturbances, has a definite advantage in finding the basic underlying cause of a problem. He can then either make a correction, or refer the patient to the appropriate doctor if the condition is not within his field, whether the treatment needed is functional or surgical.

One of the major difficulties in evaluating nerve compression has been the lack of methods for quantitatively measuring its effects. Luttgies and Gerren,³⁷ referring to this problem, state that “There exist few diagnostic maneuvers readily quantified in the literature, (to measure behavioral correlates of compression disorders) and very few innovative attempts have been aimed at remedying the situation. Until such quantification occurs, it is unlikely that the relation between compression and behavior will be thoroughly understood.” Although satisfactory quantitated muscle testing has not been developed, manual muscle testing appears to be a step in the right direction toward understanding entrapment or compression syndromes. The excellent treatment results with the easily diagnosed carpal tunnel syndrome help put this in perspective. Because of these results, it appears that there will be a giant step forward as electrophysiology is correlated with manual and quantitative muscle testing. The current procedures of applied kinesiology, using manual muscle testing, have added greatly to the clinical application of evaluation and therapeutics in cranial primary respiratory activity. Combined with the physician’s knowledge of standard neurologic examination, there is greater ability to differentiate between, and care for, pathologic and functional conditions.

Format of Cranial Nerve Presentation

The presentation of the cranial nerves in this text is not intended to be a thorough dissertation about neuroanatomy, physiology, or examination procedures. It is intended to review the course of each nerve and possible areas of entrapment in the organism. Reporting possible areas of entrapment leads to considerable speculation. The specific areas of entrapment have not been studied thoroughly, nor

has the phenomenon of subtle entrapment itself been adequately studied; consequently, there are few references in the literature to cranial peripheral nerve entrapment.^{38, 39, 40} The material in this text is designed to provide a quick reference for cranial nerve distribution, general anatomy, and standard cranial nerve examination procedures. The format of presentation is as follows.

ANATOMY AND DISTRIBUTION

General neuroanatomy^{2, 7, 8, 10, 12, 26, 50, 71} is presented in abbreviated form, with particular attention to the areas where a nerve may be encroached upon by pathology or structural distortion in the form of cranial faults, as well as some possible involvements outside the cranium. Structures innervated will be

listed, particularly when they correlate with clinical findings of involvement with the cranial mechanism. Further, neurophysiology^{5, 18, 24, 28, 56} will be discussed when applicable to the understanding of the cranial primary respiratory mechanism.

POSSIBLE ENTRAPMENT

Entrapment of cranial nerves is hypothesized to be the reason for much of the clinically observed dysfunction attributed to cranial dysfunction. Pressure on a nerve was demonstrated by Granit et al.²⁵ to create an "artificial" synapse between the motor and sensory fibers of a nerve. This cross stimulation was caused by a pressure so light that it did not interfere with the original impulse. They then removed the pressure and irrigated the nerve with a saline solution; after a brief time, the nerve returned to normal function. This seems to indicate that impingement on a nerve from cranial dysfunction is a plausible explanation for disturbances observed clinically. It remains to be demonstrated electrophysiologically that this, in reality, is what occurs in cranial dysfunction. The areas of possible entrapment are presented here as a conjecture of what might be taking place. Written material on this subject is primarily by Magoun,^{38, 39, 40} although many authors have related to the subject sporadically in papers and lectures.

A possible physiological explanation of peripheral nerve entrapment is presented by Gardner,²² whose hypothesis is based on maintenance of intercellular space. He discusses the report of Weiss and Woodbridge,⁷³ who stated that "All metazoan cells so far examined carry a negative charge at their electrokinetic surfaces." This causes the cells to have

an electrostatic repulsion since like charges repel. Their report dealt with the prevention of red blood cells from adhering to each other, and to other factors. Gardner speculated that "... in the case of brain cells, a similar repelling force would interfere with contact between adjoining cell membranes, which would explain the uniform intercellular space of 150-200 angstroms that accompany all of the intricate meanderings of the cell processes." This electrostatic repulsion would be carried throughout the nervous system, extending into the spinal cord and peripheral nerves such as the cranial and spinal nerves. The axons then would be floating and would be acted on by two opposing forces — that of the electrostatic repulsion and of the mechanical force of the surrounding tissues. Gardner goes on to point out that the interaxonal distance would be affected by the nerve going over a sharp ridge, such as the petrous portion of the temporal bone. Other mechanical disturbances may also have an effect.

Within this text the areas of peripheral nerve entrapment of the cranial nerves by cranial primary respiratory dysfunction are those which seem reasonable from study of their neuroanatomy. Until quantitative research is done to delineate the exact areas of entrapment and the mechanisms taking place, this approach will have to suffice in partially explaining the clinical results obtained.

EXAMINATION

The examination methods presented for the cranial nerves are those of classic neurology as stated in various references.^{3, 4, 14, 18, 28, 43, 44, 53, 61, 72, 78} In most cases standard neurologic examination checks the nerve for pathological changes which, if present, are not applicable to cranial fault treatment by applied kinesiology methods. If there are positive findings on standard neurologic examination, they should be closely evaluated to determine if conservative treat-

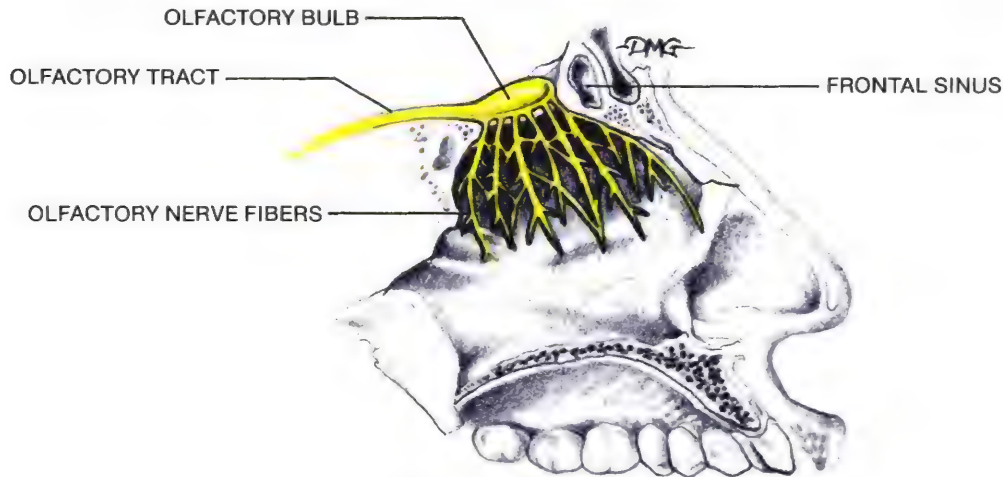
ment is applicable as a therapeutic approach. In some instances, dramatic and rapid correction of positive findings is obtained with this treatment, indicating the involvement to be functional. Again, it is important to correlate the history and clinical laboratory findings to determine the choice of treatment, and whether consultation with members of the healing arts in other specialties is necessary.

I — Olfactory Nerve

ANATOMY AND DISTRIBUTION

The olfactory nerve serves the sense of smell. Its fine sensory cells are located in specialized epithelium in the upper posterior part of the nasal cavity. They form bundles which pass through the cribriform plate of the ethmoid in two groups, ending in the olfactory bulb and resting on the intracranial surface of the

cribriform plate. The dura mater covering of the olfactory tract is continuous into the periosteum of the nose. The olfactory bulb connects with the brain via the olfactory tract which, although looking like a nerve, is more accurately classified as part of the brain.²⁶



5—1. Olfactory nerve (I).

POSSIBLE ENTRAPMENT

The olfactory nerve does not appear to have any obvious involvement with functional nerve entrapment. It is possible that distortion of the frontal bone could compress the ethmoidal notch in which the cribriform plate fits. In the adult there is apparently more limited movement of the frontal bone than

other areas of the skull. Often in older skulls there is ossification of the lower portion of the coronal suture, indicating less movement in this area. Magoun⁴⁰ attributes congestion and chronic nasal drip to narrowing of the ethmoidal notch, crowding the ethmoidal and frontal sinuses.

EXAMINATION

Prior to the test, impaired olfactory sense as a result of intranasal disease should be excluded. Loss of smell can be from nasal dryness as a result of involvement of the maxillary division of the trigeminal nerve, or from some other cause. Elderly people with chronic rhinitis often have a loss of smell, perhaps even to the point of anosmia.

The patient closes his eyes prior to the test. A substance with a strong odor — such as wintergreen, camphor, fresh coffee grounds, etc. — is placed under one nostril; the other is occluded and the

patient is asked if he can detect a scent. The test is repeated with and without an odor. If the patient perceives the odor he is asked to identify it, although this is not necessary. As long as the patient can detect the odor, anosmia can be excluded.

Anosmia, especially if unilateral, may indicate a frontal lobe tumor. Trauma may cause tearing of the nerve tissue or may create a functional problem due to cranial faults. Severe trauma involving fracture of adjacent bones may pave the way for infection of the meninges through the nasal cavity.

II — Optic Nerve

ANATOMY AND DISTRIBUTION

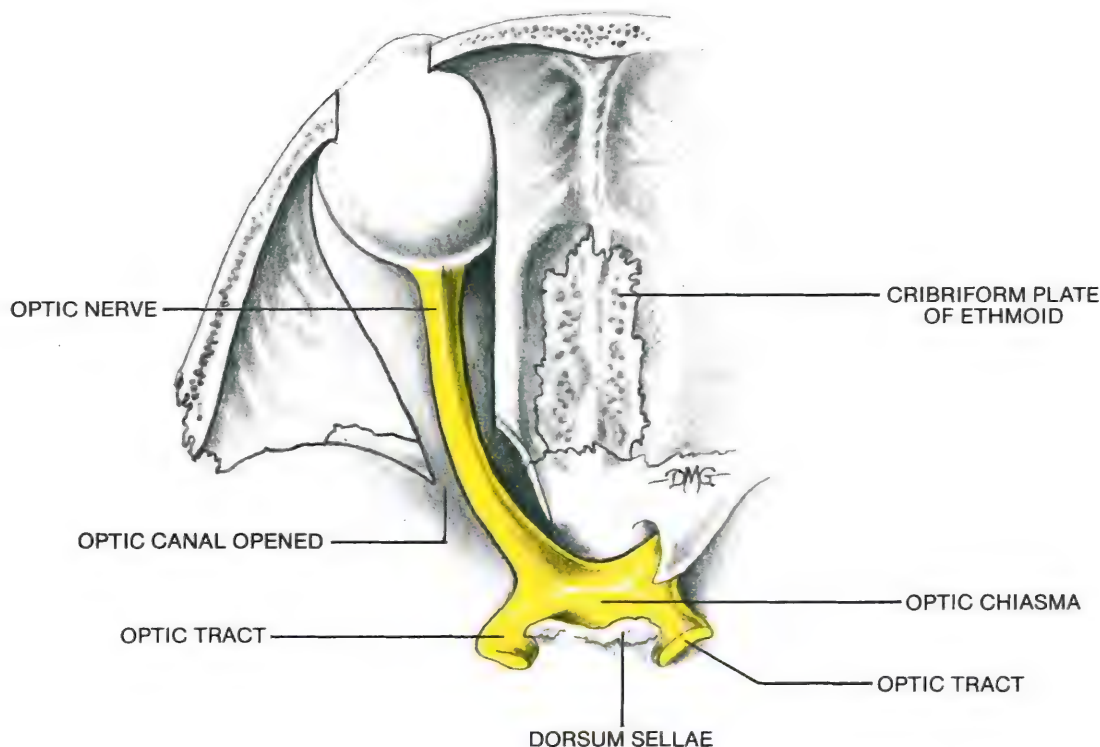
The optic nerve and the retina are developmentally part of the brain. This "nerve" is made up of the axons or central processes of the ganglion cells which lie near the surface of the retina.

The optic nerve is located in four areas: 1) the bulb of the eye; 2) the orbit; 3) the optic canal; and 4) the cranial cavity. The intraocular portion is from the ganglionic layer; it converges on the optic disc to ultimately make up bundles which collect to form the optic nerve. The orbital portion has a slightly curved course, about 6 mm longer than the distance between the optic canal and the eyeball. This is so that when turning the eye there is no restriction from the nerve length.

The optic nerve has three coverings. The outer one is derived from the dura mater and is continuous with the sclera anteriorly. The middle covering is from the arachnoid mater, and the inner from the pia mater. In the anterior portion of the orbit, the nerve

is cushioned by a quantity of fat. Toward the posterior part of the orbital portion, the nerve is in close contact with the four recti muscles. The nerve's length within the orbital portion is 20-30 mm. The nerve shares the optic canal with the ophthalmic artery. The canal is in the root of the lesser wing of the sphenoid. The intracranial portion of the nerve is about 10 mm long. It progresses posteriorly over the anterior part of the cavernous sinus and the diaphragma sellae to the optic chiasma — which rests on the tuberculum sellae of the sphenoid bone — and the diaphragma sellae of the dura. The optic tracts diverge from the posterior of the optic chiasma. The complex of the optic nerves, optic chiasma, and optic tracts makes what appears to be the letter "x."

The optic tract crosses under the cerebral peduncle, contacting the 3rd ventricle. It terminates as a medial-lateral root.



5—2. Optic nerve (II).

POSSIBLE ENTRAPMENT

In the first two sections of the optic nerve — the intraocular and orbital portions — there is significant protection of the nerve and little chance for entrapment. A vulnerable area may be where the nerve traverses the optic canal of the sphenoid. Entrap-

ment may result from the sphenoid shifting, usually as a result of trauma in the infant where more mobility is present; it could possibly occur in the adult.

EXAMINATION

There are three types of tests for the optic nerve — for visual acuity, extent of peripheral vision, and ophthalmoscopic examination of the retina and optic nerve head.

Visual acuity: Accurate evaluation of visual acuity is best accomplished by an optometrist or ophthalmologist, or others specifically trained in the field. The visual acuity evaluation described here is for neurologic purposes, and is usually done with the patient wearing his prescription glasses to correct any refractive error. This gives the physician an opportunity to evaluate any major changes in vision as a result of recent trauma, etc.

Vision is measured for close and distant function. Distant function is evaluated with a Snellen Eye Chart placed twenty feet from the patient. If the examination room is less than twenty feet, a reversed Snellen Eye Chart can be obtained and placed behind the patient. The patient looks into a mirror to see the eye chart behind him. The total distance from patient to mirror to eye chart should equal twenty feet. The eye chart is marked with the number “20” over another number. The line indicated 20/20 is normal vision; an individual can read what he should be able to at twenty feet. The numbers 20/40 indicate diminished visual acuity; a person with normal vision would be able to read that line at forty feet. The use of the time-honored Snellen Eye Chart for visual examinations should be considered as just that — an old-fashioned method for evaluating vision. It has definite limitations.

Near visual acuity is tested in a similar manner with the American Medical Association’s reading card. The card is designed to be read at fourteen inches; the lines of varying sizes are designated as “14” over another number. The line marked 14/14 indicates normal vision; an individual is able to read that line at fourteen inches.

An improved method to aid the general clinician in evaluating visual acuity is the ophthalmic telebinocular unit.⁴⁷ This is a binocular vision testing and training instrument. It permits rapid, reliable checking of basic visual patterns, and tests phorias, suppressions, binocular acuity, fusion, stereopsis, and color discrimination. The instrument uses inter-

changeable stereo targets for the various tests. There are also attachments available to test peripheral vision in a more accurate way than is usually done clinically, but not as accurately as the more specialized equipment of the optometrist or ophthalmologist. (The ophthalmic telebinocular is discussed more thoroughly in Volume V.)

Peripheral vision: The term “perimetry” designates the testing of the vision fields. This is ordinarily done with specialized equipment. When lesions are present it is an important method for determining the probable site of pathology.

The visual fields can be grossly tested by the “confrontation test.” The examiner covers one of the patient’s eyes with a card and then has the patient fix his gaze directly into the examiner’s eye. The examiner then moves his finger from outside the patient’s field of vision slowly toward it. The patient reports when the finger is first sighted. This test is done for the four quadrants of vision — the nasal and temporal, which are divided into superior and inferior.

Ophthalmoscopic examination: In addition to the evaluation of the fundus, the ophthalmoscopic examination may provide significant information about the systemic health of an individual. The ophthalmoscopic examination is out of the scope of this text. If the subject is unfamiliar, there are many texts available which give step-by-step procedures and illustrations. Most standard physical diagnosis books⁵³ cover the subject adequately for the general practitioner.

Visual field defects, swelling of the optic disc, or optic atrophy indicates that consultation with a neuro-ophthalmologist is necessary. This does not preclude evaluation of the cranial mechanism for any possible involvement.

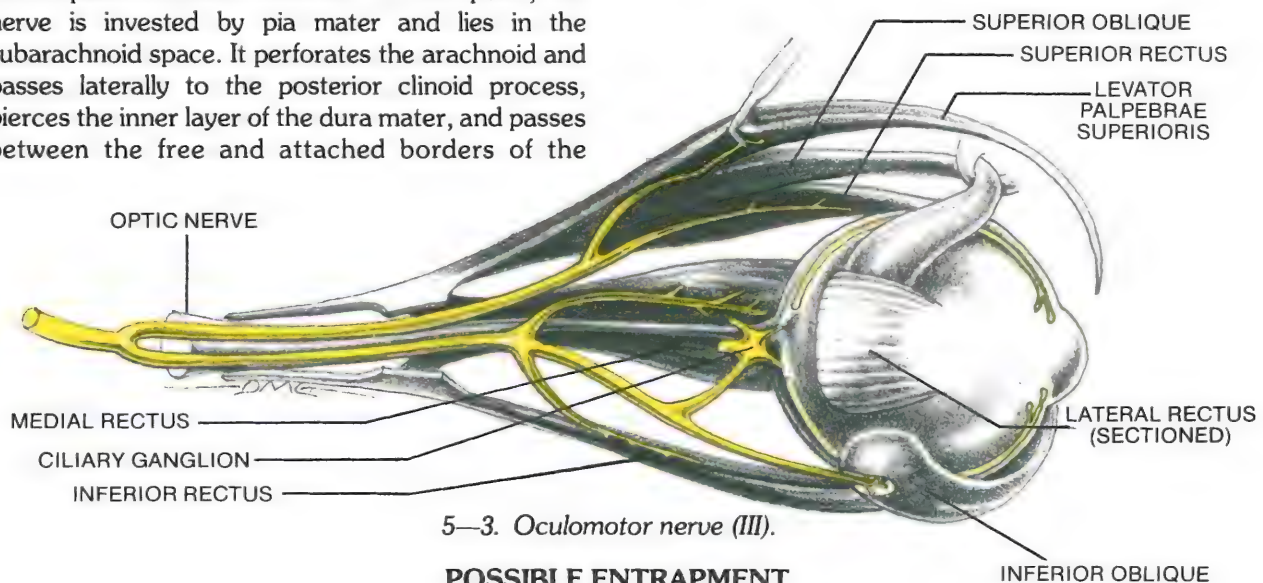
Sometimes there are dramatic changes in visual acuity following cranial fault correction. There are several mechanisms by which this improvement may take place. It is possible there may be a direct functional entrapment of the optic nerve, or there may be change in the orbital shape as a result of cranial faults (see Volume V).

III — Oculomotor Nerve

ANATOMY AND DISTRIBUTION

The oculomotor nerve is primarily a motor nerve containing special somatic efferent fibers for most of the extraocular muscles and parasympathetic fibers for the ciliary ganglion. Its superficial origin is from the midbrain at the oculomotor sulcus, emerging on the medial aspect of the cerebral peduncles near the upper border of the pons. Shortly after leaving the brain stem, it passes between the posterior cerebral and superior cerebral arteries. To this point, the nerve is invested by pia mater and lies in the subarachnoid space. It perforates the arachnoid and passes laterally to the posterior clinoid process, pierces the inner layer of the dura mater, and passes between the free and attached borders of the

tentorium cerebelli. It continues anteriorly in the outer wall of the cavernous sinus. It divides into the superior and inferior rami, which enter the superior orbital fissure to supply the medial, superior, and inferior recti muscles and the inferior oblique and levator palpebrae superioris muscles. Through the ciliary ganglion, it gives parasympathetic postganglionic fibers to the ciliaris and sphincter pupillae muscles.



Most of the areas of possible entrapment are the same areas where the nerve can be damaged as a result of trauma. These are between the somewhat rigid posterior cerebral and superior cerebral arteries as the nerve passes between the free and attached borders of the tentorium cerebelli, at the dural attachments on entering the cavernous sinus, within the cavernous sinus, and at the superior orbital fissure.

The free edge of the tentorium seems particularly to be a site of possible entrapment. Cranial faults involving the occipital, temporal, or sphenoid bones appear to change the position of this structure, thus irritating the nerve sufficiently to change its transmission characteristics.

Cranial nerves III, IV, VI, and the ophthalmic and maxillary branches of V traverse the cavernous sinus. Pressure as a result of poor drainage could possibly cause entrapment at this area, as well as torsion of the nerve by dural attachments upon entering the cavernous sinus. In studies of 2,000 cases of paralysis of cranial nerves III, IV, and VI at

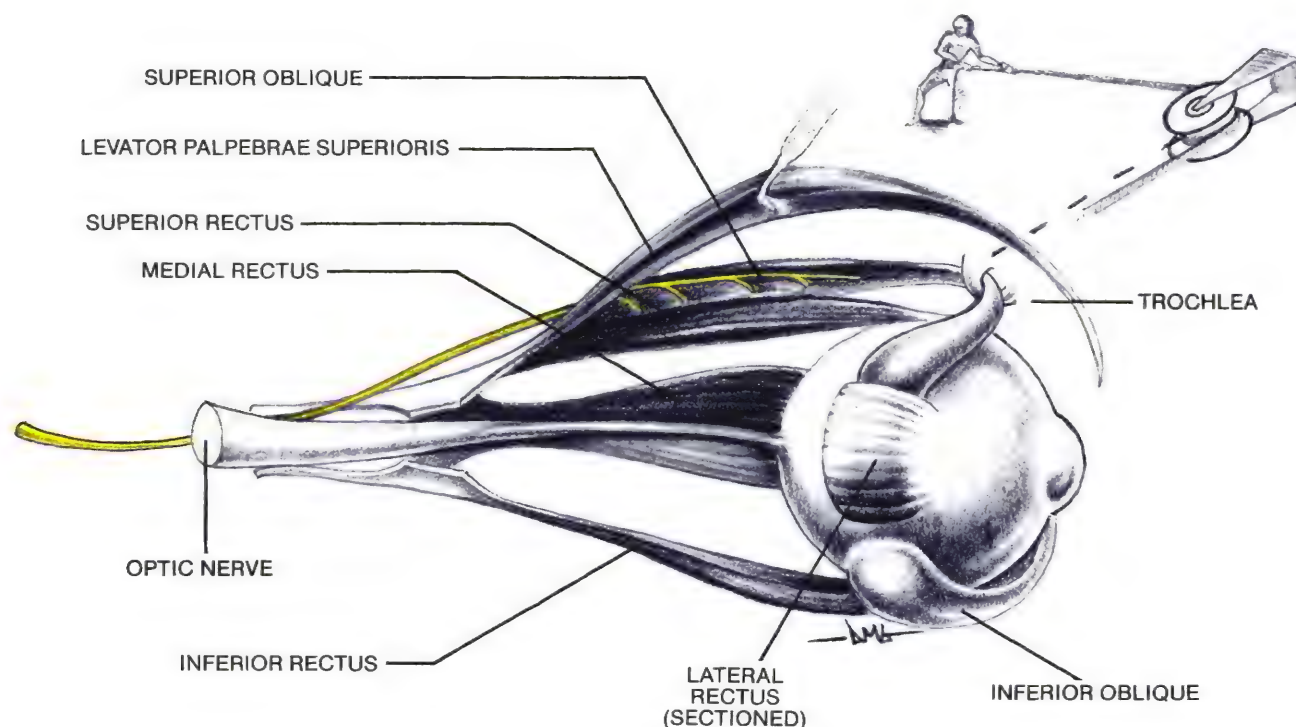
the Mayo Clinic^{57, 58} where cranial III was involved individually, the following etiology was listed after a thorough diagnostic workup: head trauma — 14%; neoplasm — 14%; vascular disease — 18%; aneurysm — 19%; and other causes — 10%. In 25% of the cases, the cause was undetermined. From these studies an observation was made regarding pupillary reactions. When the extraocular muscles supplied by cranial III are paralytic and the pupil reacts normally, the cause is probably occlusive vascular disease, especially that associated with diabetes.²³ In the 19% of cases noted above which were due to aneurysm, the reactions of the pupil were normal in only four cases. In two of these cases there was only slight indication of muscle paralysis, and the other two cases were diagnosed without arteriography. The diagnosis was presumptive. Normal reaction of pupils is a significant indication that aneurysm is not the cause of cranial III paralysis. Examination procedures for cranial nerve III are discussed after all of the other nerves dealing with the extraocular muscles (IV and VI) have been discussed.

IV — Trochlear Nerve

ANATOMY AND DISTRIBUTION

The trochlear nerve derives its name as a result of supplying the muscle which passes through a trochlea or pulley — the superior oblique muscle. This is the smallest of the cranial nerves, arising medially and posteriorly to the inferior colliculus. The nerve is directed laterally across the superior cerebellar peduncle, winding around it near the pons. It courses anteriorly along the free border of the tentorium cerebelli to pierce the dura immediately

below the border of the tentorium cerebelli, just posterior to the posterior clinoid process. It continues anteriorly through the lateral wall of the cavernous sinus. Here it is closely attached by connective tissue to the oculomotor nerve and the ophthalmic division of the trigeminal nerve. The trochlear nerve courses superiorly to enter the superior orbital fissure and to supply the superior oblique muscle.



5—4. Trochlear nerve (IV).

POSSIBLE ENTRAPMENT

The trochlear nerve is delicate in structure and has a long course. It is protected by the tentorium to the point where it pierces the dura, where nerve entrapment may develop. There may also be involvement as it enters the cavernous sinus, or as it courses through the superior orbital fissure.

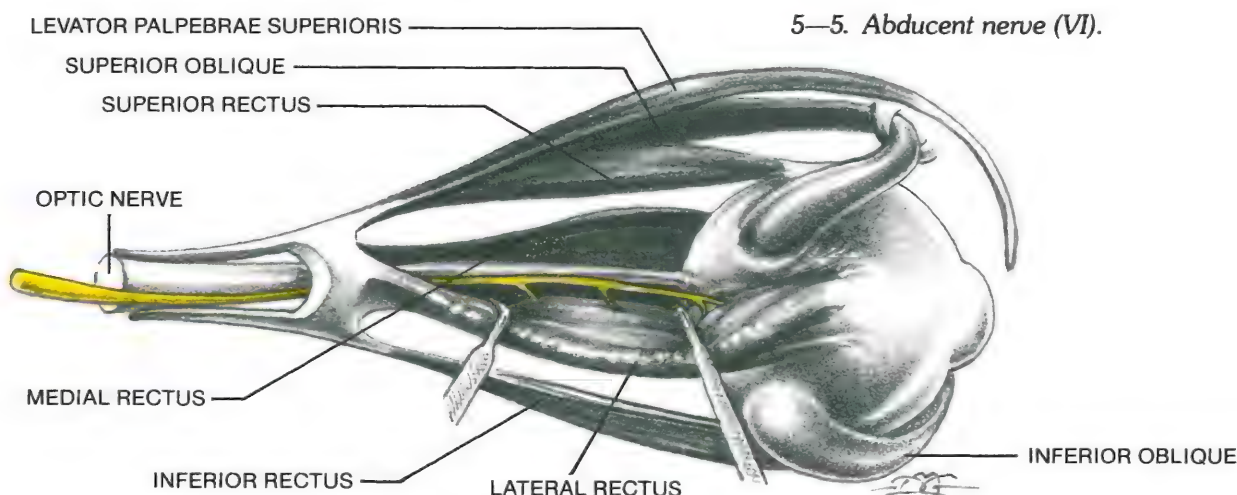
In the Mayo Clinic study previously cited^{57, 58} cranial nerve IV was individually paralyzed in 151 of the 2,000 cases. Head trauma was responsible for 31%, neoplasm for 7%, vascular disease for 24%, and other causes 13%. The cause was undetermined in 25%, and there were none due to aneurysm.

VI — Abducent Nerve

ANATOMY AND DISTRIBUTION

The abducent nerve supplies the lateral rectus muscle of the eyeball. Its superficial origin is at the sulcus between the inferior border of the pons and the medulla oblongata. It courses anteriorly, laterally, and superiorly to pierce the dura mater lateral to the dorsum sellae of the sphenoid bone. From here it

bends sharply anteriorly as it crosses the superior border of the petrous part of the temporal bone, close to its apex, being inferior to the petrosphenoidal ligament. It traverses the cavernous sinus to enter the orbital cavity through the superior orbital fissure, and supplies the lateral rectus muscle.



POSSIBLE ENTRAPMENT

Of the nerves controlling the external ocular muscles, the abducent is most frequently found to be in a state of apparent functional entrapment as observed by applied kinesiology methods. This is probably due to its long course and firm attachment as it enters the dura over the clivus, its right angle as it crosses the ridge of the petrous bone, and its close association with the rigid petrosphenoidal ligament and the lateral branches of the basilar artery. This same observation is made regarding traumatic damage to the nerve.⁶⁸ There may also be entrapment at the cavernous sinus or the superior orbital fissure. Schneider and Johnson⁶² reported cases of abducent nerve involvement resulting in paralysis of the lateral rectus muscle from hyperextension injury of the cervical spine resulting in spinal fractures. They attributed this to upward and posterior displacement of the brain, causing avulsion of the abducent nerve under the rigid petrosphenoidal ligament. They report one of the subjects had "... disalignment of her teeth." Clinical evidence from evaluating the cranial primary respiratory mechanism after the so-called "whiplash" accident indicates that cranial faults frequently develop as a result of this type of injury, and can cause an entrapment of the abducent

nerve (as well as others) of a functional nature. The change of occlusion noted above following a blow to the head indicates probable cranial bone misalignment from the trauma.

It is stated that most compression and contusion injuries of the abducent nerve heal spontaneously within two to five weeks.⁶⁸ This may be due to contusion to the nerve with resultant healing; it could be due to nerve entrapment as a result of cranial faults which the body corrects in its own self-maintaining, self-correcting manner. It appears that in most conditions where nerve entrapment is involved and the nerve has not actually been damaged, appropriate correction of the cranial dysfunction will immediately return normal function. This is borne out by clinical evidence of the elimination of diplopia immediately after cranial correction has been obtained.

In the studies by Rucker^{57, 58} previously mentioned, paralysis of cranial nerve VI was caused by head trauma in 12%, neoplasm in 26%, vascular disease in 11%, aneurysm in 4%, and other causes made up 21%. Twenty-six percent of the cases noted were of undetermined cause.

Examination of Cranial Nerves III, IV, and VI

Evaluation of cranial nerves III, IV, and VI in applied kinesiology takes two directions. First is frank paralysis which may indicate neoplasm, aneurysm, nerve disease, or direct trauma to the nerve, as well as other pathologic conditions. The second evaluation is for functional disturbances, often much less obvious but nevertheless problematic to visual function and its integration with other neurologic and body mechanisms.

The physician's first encounter with a patient during consultation gives some indication of the eyes' ability to function efficiently on a bilateral basis. Gross deviation is usually apparent. Poor eye coordination and development of dominance may cause the patient to have a "shifty-eyed look"; he continually shifts dominance from one eye to the other. This generally indicates there will be poor eye function on a bilateral basis, as observed in the "ocular lock" test of applied kinesiology (see Volume I).

When observing an individual, the examiner must be careful to actually see what he thinks he sees. In some cases, there is a bilateral skin fold at the medial aspect of the eye, called an epicanthus, which causes a widening of the bridge of the nose and gives the appearance of esotropia.

Observing a light reflection from the cornea is a simple method for determining if the eyes are looking straight at an object. The examiner directs a single

light source — such as a flashlight — toward the eyes while the subject looks at it. If there is orthophoria, the reflection from the cornea will be centered in both pupils. If there is deviation in one eye, the reflection will be off-center approximately 1 mm for each 7° of deviation.

In the cover test the examiner watches the patient's eye as the other is covered. The patient should look at a specific point while the examiner uncovers the covered eye and observes for movement, which indicates the necessity to fix the eye on the point because it was not fixed on the point with monocular vision. This is repeated with the other eye.

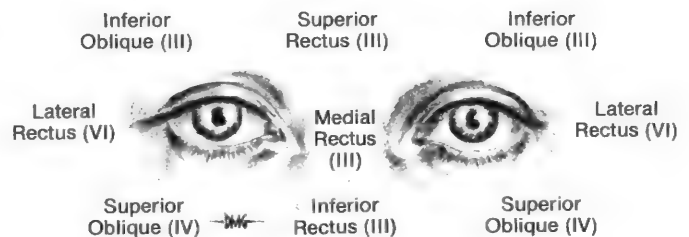
Alternately moving the cover from one eye to the other is called the alternate cover test. If the eyes have difficulty in fixing on the point, there will be a realignment in excess of 5° as the eye is uncovered. Transferring the cover to the opposite eye causes the shift to take place again. The cover test also evaluates the patient's ability to maintain his eyes in the proper position when binocular function is removed. The subject's vision is directed to a specific point; while he continues looking at that point the examiner covers one eye and observes for deviation of the covered eye when the binocular function has been removed. A deviation of up to 5° is normal for this test because the binocular fusion reflexes are eliminated.

CARDINAL POSITIONS OF GAZE

The extraocular muscles are evaluated by having the subject move in the six cardinal directions of eye movement. Each direction relates to specific muscle activity. The directions are as follows:

Lateral	—	Lateral rectus
Medial	—	Medial rectus
Medial superior	—	Superior rectus
Medial inferior	—	Inferior rectus
Lateral superior	—	Inferior oblique
Lateral inferior	—	Superior oblique

Observe that there is not a cardinal motion for directly superior or inferior. Also, the superior and inferior movements of the eye, when in a medial position, are the same as the name of the superior and inferior recti muscles; however, when in the lateral position, the superior movement is done by the inferior oblique and the inferior movement is done by the superior oblique. The motion is opposite the name of the muscle.



Limitation of motion in one of the cardinal directions of eye movement is usually due to a neurologic cause, either pathological or functional. The examiner must eliminate other possible causes of muscular dysfunction or factors which may restrict the movement of the globe. There may be disturbance of the extraocular muscle itself, as in myasthenia gravis, exophthalmic goiter, or Duane's retraction syndrome (a congenital deficiency of abduction). There may be mechanical obstruction from a tumor or inflammation.

When there is a gross defect in moving into the cardinal positions, it is easy to determine which muscle is involved. When moving into the cardinal positions shows no gross change, yet there is diplopia, it becomes necessary to determine which muscle(s) and which eye are involved.

A simple procedure for determining which eye is giving the false image is to have the patient look through a red lens over one eye and a green lens over the other. This lens arrangement is called an anaglyph. A string is tied to the nasal piece of the frame holding the lenses, and a small ball is attached to the other end. The examiner holds the ball approximately six feet from the patient. The patient directs his vision to the ball. There will appear to be two strings, both normally leading to the ball. Each eye independently views the string. The string will take on the color of the lens through which it is viewed; for example, if the red lens is over the right eye, the image viewed by the right eye will be an apparently red string, while the other eye will perceive a green string. In diplopia both strings will not converge into the ball; one string will lead to the ball, and the other will deviate. The string perceived as deviating may go in any direction. The strings may appear to cross, fail to meet, or deviate up or down. The color of the string which the patient sees going to the ball indicates which eye has the true image.

Lateral or vertical phoria can be tested more accurately in a clinical situation with an ophthalmic telebinocular. With the anaglyph or ophthalmic tele-



5—6. Anaglyph test.

binocular, it is important to test an individual in various positions. There may be normal convergence of the eyes when the neck and head are in a neutral position; phoria may develop when the cervical spine is extended, flexed, or laterally tilted. This may be from varied muscular pull into the cranium, cervical disturbances, or other factors.

When there is involvement of cranial nerves III, IV, and VI, it is possible that trauma has caused contusion, tearing, or traction on the nerve; these may not be compatible with the therapeutic efforts described in this text relating with the cranial primary respiratory mechanism. There is also the possibility of neoplasms, vascular disease and aneurysms, infections, and toxins; these must be considered by the physician during the examination for cranial primary respiratory dysfunction.

Cranial Nerve III: Complete division of the oculomotor nerve leads to paralysis of all the muscles involved; thus there is ptosis of the upper eyelid due to paralysis of the levator palpebrae superioris. The eye will deviate laterally and slightly downward from lack of opposition to the lateral rectus and superior oblique muscles (these are the only extraocular muscles which are not supplied by the oculomotor nerve). There will be pupil dilation from paralysis of the sphincter pupillae, and lack of ability of accommodation from paralysis of the ciliaris muscle.

Cranial Nerve IV: The trochlear nerve supplies only the superior oblique muscle; consequently, paralysis associated with it produces an unusual type of diplopia. As long as vision is kept above the horizontal, there is no diplopia; however, as soon as it drops below that line, diplopia develops.

Cranial Nerve VI: With complete separation of the abducent nerve, the eye deviates medially from lack of opposition of the lateral rectus muscle.

Functional disturbance of cranial nerves III, IV, and VI is most often observed in applied kinesiology by testing for ocular lock. The test may be done by having the patient look into the cardinal positions of gaze; the examiner tests a previously strong indicator muscle for weakening while the patient maintains the eye position. A positive indication of poor function of these cranial nerves is the weakening of the indicator muscle as observed by manual muscle testing. A dynamic method of evaluating for ocular lock is to have the patient follow the examiner's finger in a circular motion and then test a previously strong indicator muscle. This is done in both clockwise and counter-clockwise directions. Weakening will usually be in only one direction if the test is positive. After correction of cranial faults, the ocular lock test will be negative, indicating improved eye coordination.

V — Trigeminal Nerve

ANATOMY AND DISTRIBUTION

The trigeminal nerve is the largest of the cranial nerves. It is the sensory nerve of the face, most of the scalp, the teeth, the mouth, and the nasal cavity. It is the motor nerve of the muscles of mastication and some additional muscles. The trigeminal nerve contains proprioceptive fibers from the masticatory muscles and possibly from the extraocular muscles. Its superficial origin is near the upper border of the ventral surface of the pons. There is a large sensory and small motor root.

The semilunar ganglion (trigeminal, Gasserian) is in a recess in the dura mater, covering the trigeminal impression near the apex of the petrous portion of the temporal bone. The ganglion is lateral to the posterior part of the cavernous sinus and the internal carotid artery at the foramen lacerum. The sensory fibers are continuous from the semilunar ganglion to their attachment at the pons. The motor root passes between the ganglion and the petrous portion of the temporal bone and leaves the skull through the foramen ovale.

The ganglion is made up of contributions from three large nerves — the ophthalmic, the maxillary, and the mandibular. The ganglion also receives filaments from the carotid plexus of the sympathetic, and gives off small twigs to the tentorium cerebelli and to the dura mater of the middle cranial fossa.

Ophthalmic Nerve

The ophthalmic nerve is the superior division of the trigeminal, and is entirely sensory. It supplies branches to the eyeball, conjunctiva, lacrimal gland, part of the mucous membrane of the nose and paranasal sinuses, skin of the forehead, scalp, eyelids, and nose.

Arising from the semilunar ganglion, the ophthalmic nerve courses anteriorly in the cavernous sinus close to the lateral wall, dividing into three branches — the lacrimal, frontal, and nasociliary — prior to entering the orbit through the superior orbital fissure.

The lacrimal nerve enters the orbit through the lateral part of the superior orbital fissure, which is its narrowest part. It supplies the lacrimal gland and the adjacent conjunctiva. Whether it contains secretory fibers for the gland is controversial.

The frontal branch — the largest branch of the ophthalmic — is primarily the continuation of the nerve. It enters the orbit through the superior orbital fissure, coursing anteriorly between the levator

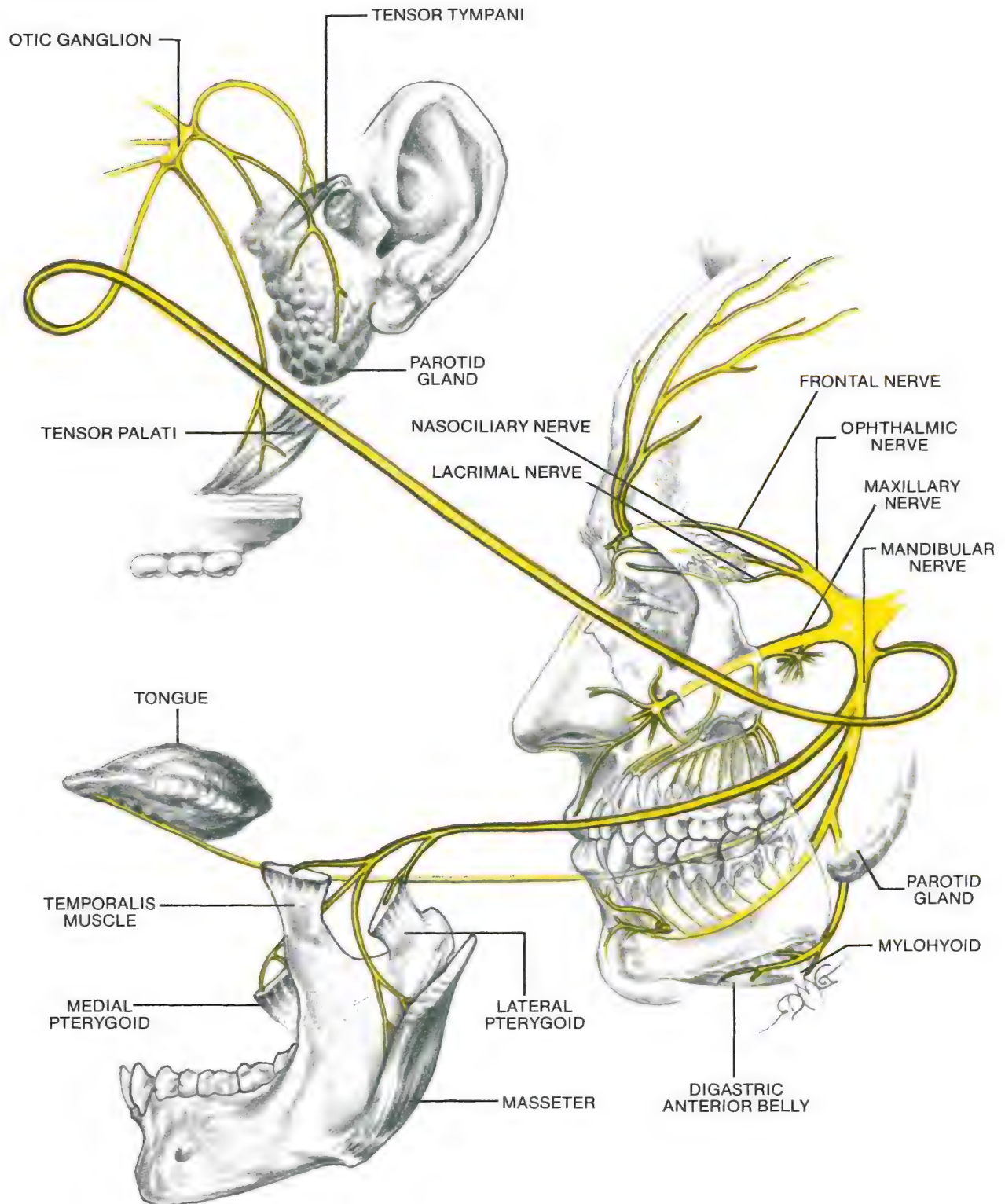
palpebrae superioris and the periosteum. Midway into the orbit it divides into a small supratrochlear and large supraorbital branch. The supratrochlear nerve gives off small branches and pierces the orbital fascia to give filaments to the conjunctiva, skin of the medial part of the upper lid, and skin of the lower and middle parts of the forehead. The supraorbital nerve leaves through the supraorbital notch, supplying branches to the upper eyelid, scalp, and mucous membrane of the frontal sinus.

The nasociliary nerve enters the orbit through the medial part of the superior orbital fissure between the two heads of the lateral rectus, and between the superior and inferior divisions of the oculomotor nerve. It passes through the medial wall of the orbital cavity as the anterior ethmoidal nerve, entering the cranial cavity superior to the cribriform plate of the ethmoid bone. It enters the nasal cavity through a slit at the side of the crista galli to supply branches to the mucous membrane of the nasal cavity. The nasociliary nerve then emerges from between the inferior border of the nasal bone and the lateral nasal cartilage under the name external nasal branch; it supplies the skin of the ala, apex, and vestibule of the nose. The long ciliary nerves — which supply the iris and the cornea and probably provide sympathetic fibers to the dilator pupillae muscle — are branches of the nasociliary. The infratrochlear branch supplies the skin of the eyelids and sides of the nose, the conjunctiva, lacrimal sac, and caruncula lacrimalis. The ethmoidal branches supply the mucous membranes of the ethmoidal and sphenoidal sinuses. The internal nasal branches supply the anterior part of the septum and lateral wall of the nasal cavity.

Maxillary Nerve

The maxillary nerve arises from the middle of the semilunar ganglion and is entirely sensory. It supplies the skin of the central portion of the face, nose, and upper lip, the mucous membrane of the nasopharynx, maxillary sinus, roof of the mouth, and the upper dental arch.

From the semilunar ganglion the maxillary nerve passes forward in the cavernous sinus, beneath the dura and through the foramen rotundum, leaving the cranial cavity. It then crosses the pterygopalatine fossa over the posterior surface of the maxilla, entering the orbit through the inferior orbital fissure. Its name is now the infraorbital nerve. Passing over



5—7. Trigeminal nerve (V).

Cranial Nerves

the floor of the orbit, it exits through the infraorbital foramen.

There are four groups of branches of the maxillary nerve; they are located in the cranium, in the pterygopalatine fossa, in the infraorbital canal, and on the face.

Cranial Branch. There is one branch in the cranium — the middle meningeal branch — which supplies the dura mater of the middle cranial fossa.

Pterygopalatine Fossa Branches. In the pterygopalatine fossa, the major branches are the zygomatic nerve, the pterygopalatine nerves, and the posterior superior alveolar branches. The zygomatic nerve divides into two branches — the zygomaticotemporal and the zygomaticofacial. The zygomaticotemporal branch supplies the skin of the side of the forehead and communicates with other nerves. The zygomaticofacial branch supplies the skin over the zygomatic bone and joins other nerves.

The pterygopalatine nerves can be divided into four groups — the orbital, palatine, posterior superior nasal, and pharyngeal. The orbital branches enter the orbit via the inferior orbital fissure and supply the periosteum. Filaments also supply the mucous membranes of the ethmoidal and sphenoidal sinuses.

The greater palatine nerve passes through the greater palatine canal to emerge on the hard palate through the greater palatine foramen. It supplies the gums and the mucous membrane of the hard palate. It has several branches which exit along the course of the nerve and supply the nasal cavity, soft palate, uvula and tonsil, posterior ethmoidal sinuses, and the nasal part of the pharynx posterior to the auditory tube. Further, there is supply to the gums and mucous membranes of the cheek, maxillary sinus, alveoli, and molar teeth.

Infraorbital Canal Branches. Branches given off in this area supply the alveolar sockets and teeth, other than the three molars. There is a branch which supplies the inferior meatus and the floor of the nasal cavity.

Facial Branches. These branches supply the skin and conjunctiva of the lower eyelid, skin of the side of the nose and upper lip, and mucous membranes of the mouth and labial glands.

Mandibular Nerve

The mandibular nerve is the only branch of the trigeminal that is mixed. The sensory root arises from the semilunar ganglion, while the motor root passes inferiorly between the ganglion and the petrous portion of the temporal bone. The sensory and motor parts run parallel to exit from the skull through the foramen ovale, joining just outside the skull. The main trunk is short, only 2 or 3 mm; it divides into a smaller anterior and larger posterior division.

The main trunk has two major branches — the ramus meningeus, which re-enters the skull through the foramen spinosum to supply the dura, and the mucous membrane of the mastoid air cells. The medial pterygoid nerve supplies the internal pterygoid, the tensor veli palatini, and the tensor tympani.

The anterior division of the mandibular nerve is primarily motor. It supplies the muscles of mastication and skin and mucous membrane of the cheek. The masseteric nerve supplies the masseter and gives a filament to the temporomandibular joint. The deep temporal and lateral pterygoid nerves supply the anterior and posterior portions of the temporal muscle and the lateral pterygoid muscle, respectively. The buccal nerve supplies the skin of the cheek and mucous membranes of the mouth and part of the gums.

The posterior division is reversed from the anterior in that it is primarily sensory, with a small amount of motor fibers. The auriculotemporal branch has sensory fibers which join with the zygomatic, buccal, and mandibular branches of the facial nerve, supplying the skin of these areas. There is communication with the otic ganglion to supply the parotid gland with secretomotor fibers. Other structures receive supply by nerves which course through the otic ganglion but do not synapse. The mandibular nerve also has branches supplying the skin of the ear, the external auditory meatus, temporomandibular joint, and skin over the temporal region. The lingual nerve communicates with other nerves and supplies the mucous membrane of the anterior two-thirds of the tongue, adjacent mouth and gums, and the sublingual gland. The taste buds on the anterior two-thirds of the tongue are supplied by the fibers communicated through the chorda tympani of the facial nerve. The inferior alveolar nerve supplies the mylohyoid and the anterior belly of the digastric muscle. There are also branches communicating with branches of the facial nerve, which supply the teeth of the lower dental arch and the skin and mucous membrane of the chin and lower lip.

POSSIBLE ENTRAPMENT

The semilunar ganglion appears to be in a vulnerable position for entrapment neuropathy. It lies close to the apex of the petrous portion of the temporal bone in a pocket of dura mater called Meckel's cave. Students of the cranial primary respiratory mechanism recognize that this area is one which has considerable motion within the confines of the limited motion of skull articulations. The sphenopetrous articulation appears to maintain this motion throughout life. Distortions in this area appear to cause strain on the dura, resulting in irritation to the semilunar ganglion.

Lee³⁶ first called attention to the course of the trigeminal nerve over the sharp angle of the petrous bone and proposed the hypothesis that there could be entrapment of the nerve between the sharp ridge and the superior petrosal sinus above. The possibility of entrapment neuropathy was also considered by Taarnhoj.⁶⁵ He speculated that entrapment could develop through the narrow channel formed by the dura to the upper sharp margin of the petrous bone. At this location, "Small changes either in the dura or adjacent tissues perhaps of vascular origin, could be assumed to narrow the channel so much that a compression takes place." Surgery to perform decompression at this area has been successful.⁶⁶

An x-ray study which measured the balance between the right and left portions of the temporal bones was done by Gardner.²² It was observed that trigeminal neuralgia occurred three times more often on the side of the high petrous apex than on the low side. Gardner concludes, "The most common cause of trigeminal neuralgia is compression of the nerve root by an aberrant branch of the superior cerebellar artery or by angulation over the petrous apex. This angulation is due to the fact that in passing forward the nerve root pursues an upward course indicating that the petrous apex is higher than nature intended, in other words that the base of the skull is pushed in." Gardner relates this basilar impression to be a human trait resulting from the force of gravity exerted on the relatively heavy head because of the upright posture. Viewing this in the cranial primary respiratory concept, the petrous portion of the temporal bone is a highly mobile area, especially at the apex. Selby⁶³ questions any correlation of trigeminal neuralgia with the elevation of the petrous portion as observed by Gardner, since similar asymmetry is seen in patients who have not had trigeminal neuralgia. Again, relating this to the cranial primary respiratory concept, it is observed that all individuals who have asymmetry do not have cranial faults; in other words, there is not necessarily

disturbed neurologic function or cranial movement because of lack of symmetry.

Malis⁴² finds a constant dural band of fibers crossing the posterior root at the petrous apex. He suggests that "... in trigeminal neuralgia there is a mechanical lesion that produces a derangement of axons in the posterior root at a point of angulation or compression. The point of derangement may be at the petrous pyramid where increasing elevation of the pyramid with age alters the angle of the dural band and narrows the opening. ..." Surgical decompression of this area is usually successful in relieving the pain of trigeminal neuralgia. This seems to indicate that loss of functional motion of the temporal bone may interfere with normal nerve activity at this location.

There can be entrapment of the ophthalmic nerve as it courses through the lateral wall of the cavernous sinus, or at the superior orbital fissure. Entrapment at the fissure can result from dural tension or possibly from an actual shift of bones, narrowing the opening. The lacrimal branch enters through the narrowest portion of the fascia. The ethmoidal branch of the nasociliary nerve traverses a foramen between the ethmoid and frontal bones. The structure of the ethmoid at this area is very thin, offering the possibility of easy narrowing of the foramen.

The maxillary branch of the semilunar ganglion progresses into the cavernous sinus, through the foramen rotundum, through the pterygopalatine fossa, and traverses the inferior orbital fissure. Dural tension at the foramen rotundum, or a shift in the shape of the inferior orbital fissure, may cause entrapment.

The mandibular nerve joins its two roots just after the sensory root arises from the semilunar ganglion, the motor root having passed inferiorly between the ganglion and the petrous portion of the temporal bone. Possible entrapment of the motor root may occur at this area. The mandibular nerve may become entrapped at the foramen ovale as it exits the skull. There may also be entrapment from the considerable fascial investment of the nerve in relation to the pterygoid muscles and fascial attachments on the basilar surface of the skull.¹¹

Pathological conditions involving the semilunar ganglion are infection with herpes zoster virus, primary neurinoma, and neoplasms from adnexa.⁶³ Nerve entrapment of all branches of the trigeminal nerve can be caused by neoplasms and vascular disorders. Occasionally there is numbness preceding severe pain. Harris²⁹ evaluated the possibility that

Cranial Nerves

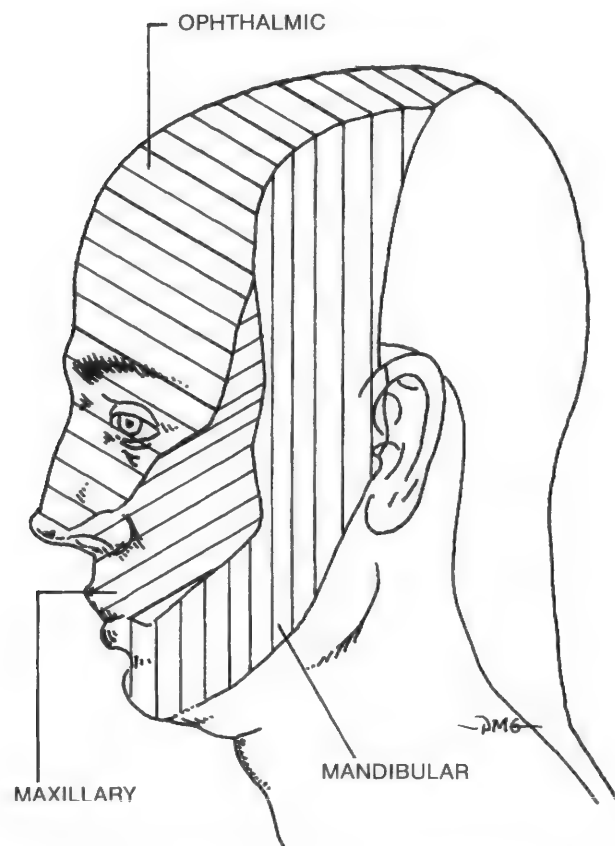
trigeminal neuritis is due to latent disseminated sclerosis, and rejected the hypothesis.

Trauma can damage the nerve. Probably trauma that causes damage to the nerve and trauma which causes cranial faults and subsequent entrapment

neuropathy are very similar, differing only in magnitude and the uniqueness of each injurious episode. This requires the physician to be constantly alert for subdural hemorrhage and direct trauma to the nerve tissues.

EXAMINATION

The divisions of the trigeminal nerve are examined for sensation by the usual methods: wisps of cotton, pin pricks, and thermal sensation. Overlap of the dermatomes of the ophthalmic, maxillary, and mandibular divisions is very limited as opposed to other areas of the body. Illustration 5—8 pictorially shows the areas of sensation by the various branches.

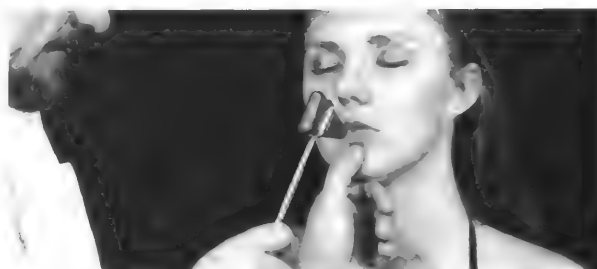


5—8. Trigeminal nerve sensory distribution.

The ophthalmic division can be evaluated by the corneal reflex test. The cornea is lightly touched with a tuft of damp cotton which has been twisted into a point. The examiner approaches the eye from the side to avoid initiating blinking as a defensive reflex of the patient. The normal corneal reflex is a quick blink of the eye. The two eyes are compared. This test

evaluates the sensory supply to the eye; the motor supply to close the eyelid is from the facial nerve.

Evaluation of the deep tendon reflex for the muscles of mastication can be accomplished in two ways, each with the patient in a seated position. (1) With the patient's mouth slightly open (1-2 cm), the examiner places his thumb on the anterior mandible just above the mental protuberance, applying pressure to open the jaws a bit wider. The examiner then uses a reflex hammer to tap his thumb, which induces the muscle stretch. Diminished reflex activity may indicate lower motor neuron involvement, while a hyperactive reflex possibly indicates an upper motor neuron lesion. (2) The patient lightly holds a tongue depressor between the molar teeth, while the examiner flicks the end of the depressor with his finger or strikes it with a reflex hammer. Observation is made for hyperactive reflex of the muscles of mastication. This is best accomplished with the patient's eyes closed so there is no anticipation of the flicking of the tongue depressor. The first jaw reflex test described here usually works better because it provides the ability to evaluate for an over- or underactive reflex.



5—9. Deep tendon reflex.

Direct muscle strength evaluation is difficult to obtain because of the tremendous power of the muscles of mastication. In cases of extreme weakness, the examiner may be able to open the mouth by stabilizing the head and applying force to the mental protuberance. This evaluation is not indicated in most cases. The best information can usually be obtained by observing the mandible as the patient opens his mouth. There will be deviation of the jaw to the side of weakness if there is weakness of the external pterygoid (a jaw-opening muscle). There are many additional methods for testing muscular balance and function within the framework of applied kinesiology evaluation of the temporomandibular joint and its associated muscles (refer to Chapter 14).

Trigeminal neuralgia (tic douloureux)

The treatment of trigeminal neuralgia by cranial fault correction has clinically been effective. The results are much improved if the condition is diagnosed and treated early. This is apparently due to histologic changes developing in the nerve from the entrapment. If the condition is post-surgical or post-alcohol injection, the prognosis is guarded. Although clinical results have been good, it must be pointed out that many cases of trigeminal neuralgia develop remissions and exacerbations in the absence of any treatment. It is very easy for symptomatic relief to be erroneously attributed to the treatment being given, rather than to a spontaneous remission. Before the efficacy of cranial treatment for trigeminal neuralgia can be evaluated, controlled studies with statistical evaluation need to be completed. Unfortunately, most doctors using applied kinesiology evaluation of the primary respiratory mechanism in a general practice do not see an adequate number of trigeminal neuralgia cases to conduct this type of study. This dearth of cases is realistic, since an indirect epidemiologic study by Penman⁴⁹ estimated the presence of trigeminal neuralgia is about 155 cases per million. The osteopathic approach and results reported by Lay³⁵ and Wilson⁷⁵ appear to parallel the findings in AK.

Trigeminal neuralgia is classified as idiopathic or symptomatic. Idiopathic trigeminal neuralgia is the genuine type; the pain is unassociated with any identifiable structural disease of the nervous system. The condition has a specific pattern of paroxysms of pain which is "stabbing," "lightning-like," severe shooting pain. The pain is usually generated by stimulation of a trigger zone around the lips and the mouth, by talking or eating, or by brushing the teeth or shaving. The sharpness of the pain lasts only a few seconds or slightly longer; following pain reduction

there is a refractory period in which the stimulation will not cause another pain. In some cases, the pains are so severe and relatively close together that the patient reports they do not subside for an hour or more. Interestingly, there are rarely paroxysms of pain during sleep. Routine medical treatment is carbamazepine as a conservative approach, with alcohol injection, sectioning of the nerve root, or decompression as surgical procedures.

Idiopathic trigeminal neuralgia must be differentiated from the symptomatic type. In symptomatic trigeminal neuralgia, there is actual nerve pathology requiring consultation with a neurologist or neurosurgeon for further differentiation and possible treatment or surgery. Typically, the history of the condition provides significant information to make the diagnosis. During neurologic evaluation, any abnormal physical signs are evidence to seriously consider symptomatic trigeminal neuralgia. A diminished corneal reflex or sensory loss in any of the three divisions contributes to this suspicion.

Many cases classified as trigeminal neuralgia can be effectively treated by various dental therapeutics. Some of these approaches may deal directly with pathology, while others deal with occlusion, temporomandibular joint dysfunction, and, indirectly, the cranial mechanism. Dawson¹⁶ states that in his "... clinical experience tic douloureux is almost always a misdiagnosis. It is usually nothing more than a classic TMJ syndrome and can be resolved by occlusal therapy. A number of these patients have been found to have a TMJ syndrome in combination with abscessed or 'dying' pulps or split teeth." When evaluating facial pain, it is important to obtain a history of the patient's dental health and any dental procedures that may have been performed. It is possible that the trigeminal neuralgia relates to an extraction or some other procedure.⁷⁵ There may be a traumatic neuroma of the oral cavity which relates to pain in the trigeminal nerve. The pathology is a cavity in the bone, usually at the site of a previous tooth extraction or other surgical intervention. The cavity is not visible on x-ray, but usually contains an infectious process. Treatment is to curette the area, followed by antibiotic therapy.^{54, 55, 64} When a traumatic neuroma is suspected, it is good procedure to evaluate for a "neurologic tooth" (see Chapter 9). If present, its treatment often relieves the pain without the condition recurring unless there is some other contributing factor.

It is possible for facial pain to be from remote causes. Norfolk⁴⁶ reports cases of facial neuralgia which respond to cervical manipulation. These will nearly always be in combination with cranial faults.

VII — Facial Nerve

ANATOMY AND DISTRIBUTION

The facial nerve arises superficially from two roots at the caudal border of the pons. The larger is the motor root; the smaller is sensory and called the *nervus intermedius*. Nerves from the two roots course within a sheath of dura — either as a single or double nerve — anteriorly and laterally in the posterior fossa, to enter the internal acoustic meatus. Separating from the acoustic nerve with which it traveled, the facial nerve enters the petrous portion of the temporal bone through the facial canal, running a tortuous course to exit at the stylomastoid foramen. After running through the parotid gland and crossing the external carotid artery, it divides at the posterior border of the ramus of the mandible into two primary branches — the temporofacial and the cervicofacial. From these branches there is wide distribution over the head, face, and upper part of the neck supplying the superficial muscles — primarily those of expression — and the muscles of

the scalp and external ear. The buccinator, platysma, stapedius, stylohyoid, and the posterior belly of the digastric muscle are also supplied by these branches.

The sensory portion of the facial nerve is known as the *chorda tympani*. It arises from the facial nerve just prior to its emerging from the stylomastoid foramen. The *chorda tympani* nerve emerges from the petrous portion of the temporal bone after traveling in its own canal, to have communication with the otic ganglion; it joins with the lingual nerve. The *chorda tympani* supplies the anterior two-thirds of the tongue with visceral afference for taste. It also contains some pre-ganglionic parasympathetic fibers which are secretomotor in nature. These fibers innervate the lacrimal, salivary, and other glands, and also the vessels of the mucous membrane of the palate, nasal pharynx, and nasal cavity. The facial nerve communicates with many other nerves, having a wide range of influence.

POSSIBLE ENTRAPMENT

Traction on the dural membrane from cranial faults can possibly cause entrapment as the facial nerve enters the internal acoustic meatus with the acoustic nerve. The nerve and other tissue completely fill the facial canal, leaving no room for its increase in diameter from inflammatory processes. If such should occur, a compression of the blood vessels in the canal is required.³¹ This appears to be a potential area of entrapment because of functional disturbance of the cranial primary respiratory mechanism. If there is traction on the nerve as it enters the internal acoustic meatus, it seems reasonable that the nerve would become inflamed within the canal, causing an increased amount of entrapment over

that of the initial dural irritation. Cathie¹¹ comments on the fascial investment that occurs from the close relationship of the nerve with the parotid gland and the styloid process as it emerges from the stylomastoid foramen. Strain on the fascia from many structural imbalances could cause an entrapment neuropathy here. Additional entrapment may be possible in the more peripheral nerve structure. The greater petrosal nerve exits the petrous portion of the temporal bone by way of the hiatus of the facial canal. It courses beneath the dura and the trigeminal ganglion to unite with the deep petrosal nerve; together they make up the vidian nerve which traverses the pterygoid canal (vidian canal).

EXAMINATION

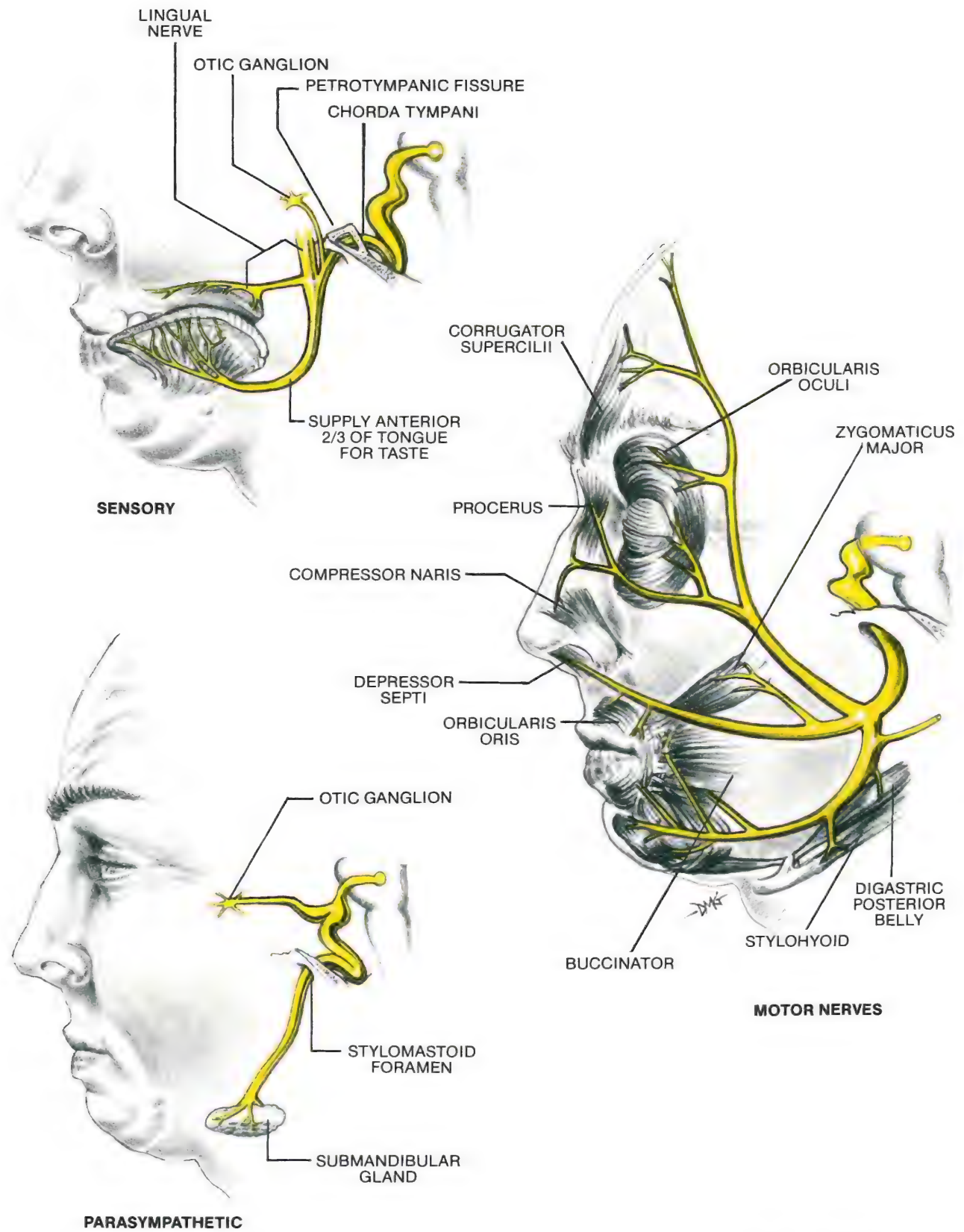
The object of the examination is primarily to differentiate "Bell's palsy," considered to be an idiopathic facial paralysis, from those conditions requiring special procedures, as in neoplasms.

Evaluation of the facial muscles begins with the physician's observation during consultation. Asymmetry is easily recognized in the muscles of facial expression as the lips failing to curl during a smile, imbalance of wrinkles in the forehead, closure of the

eyes, imbalance of the nasolabial fold, etc. Specific facial movements are described to evaluate the muscles in Chapter 4.

Facial muscles can be more thoroughly evaluated by electrodiagnosis. The types of tests are reaction of degeneration, intensity-duration curve, electromyogram, nerve conduction, nerve excitability threshold, and maximal action potential.³¹

A lesion of the facial nerve peripheral to the



5—10. Facial Nerve (VII).

Cranial Nerves

chorda tympani branching off, which is just prior to the facial nerve emerging from the stylomastoid foramen, will not cause a loss of taste. Taste can be examined with sugar, salt, sour, and bitter substances. The substance is applied in a fluid state, with a cotton applicator, to the anterior two-thirds of one side of the tongue. The patient is asked to identify the substance without drawing his tongue into his mouth; this would intermingle the substance with the opposite side or the posterior one-third of the tongue, thus confusing the test.

There is usually a lack of sophistication in this type of testing. Often there is no standard method of quantification. The use of too much fluid may allow the substance to spread to an area which the examiner does not want evaluated. This easy spreading over the already moist tongue can be observed by using colored fluid. A refined technique of testing taste with solutions has been developed by Bornstein⁶ and modified by Krarup³⁴. It describes quantitated solutions and specific procedures for reproducibility.

An improved method of evaluating taste has been developed by Krarup.^{33, 34} When anodal galvanic stimulation is given to the tongue, it is perceived to have an acidic or metallic taste. Specialized test equipment gives a variable galvanic current for the observation of its perception. Localization is good and quantification can be done by varying the current. Taste should be balanced on the two sides.

Specialized techniques are used to measure the production of tears from the lacrimal gland and saliva from the submandibular gland. Evaluation of the other cranial nerves will often indicate a space-occupying lesion. An intrapontine lesion causing facial weakness will usually also affect abducent nerve function.⁴ By quantitatively evaluating all the functions of the facial nerve, the area of lesion can usually be determined. When the facial nerve leaves

the pons, it carries fibers for motor activity, salivation, lacrimation, and taste. Fibers to the lacrimal glands branch off first, and then the salivary and taste fibers. Only motor fibers to the face emerge from the stylomastoid foramen. This evaluation has several considerations which must be made;³¹ it requires specialized equipment not usually found in a general practitioner's office.

The usual treatment for Bell's palsy (idiopathic) is corticotropin or steroid medication. The value of surgical decompression is questionable; several otologic surgeons have given up the procedure for treating the condition.^{31, 67}

The recovery of patients with Bell's palsy is usually within a short period of time and is complete, but it may be delayed and incomplete. Taverner⁶⁷ found that about 50% of the patients showed improvement in an average of ten days and completely recovered in an average of forty-five days. The slower group showed improvement in an average of about two months; they stabilized in approximately nine months, with more residual problems. These two groupings are generalized with a notation that the making of the two groups is an average of the two extremes; actually there is a continuum between the groups.

There is also a relationship of the return of muscle movement with the degree of recovery.⁶⁷ When movement returns within one month of the onset of the paralysis the mean recovery is 78%; between one and two months the mean recovery is 40%. No movement until the third month yields a mean recovery of only 28%.

The amount of recovery depends on whether the involvement is due to conduction block or whether there is destruction of the nerve fibers causing denervation. Denervation is examined for with electromyography and is found to occur in 40% of cases.⁶⁷

ABERRANT FUNCTION

There are several conditions which may become manifest as a result of abnormal function of the facial nerve. These often develop following an episode of Bell's palsy. There is the possibility of unintentional muscle activity — called synkinesis — accompanying a volitional movement of the face. One possibility of the cause of synkinesis is that during regeneration of axons following an injury to the nerve, there is an intermingling of these axons which misdirects impulses. Another possibility also exists at the site of nerve injury. Improper impulses may develop at the

site of injury and produce motor activity incompatible with the body's needs. Either cause of improper impulses can produce muscle activity, such as spasmodic contracture or random activity.

Gardner²² states that "Hemifacial spasm is the 7th nerve counterpart of trigeminal neuralgia. Each may be considered the equivalent of a focal seizure at the nerve root level." On the other hand, Norfolk⁴⁶ gives a rationale of how cervical lesions, especially of the upper cervical region, can adversely influence the facial nerve.

VIII — Acoustic Nerve (Vestibulocochlear Nerve)

ANATOMY AND DISTRIBUTION

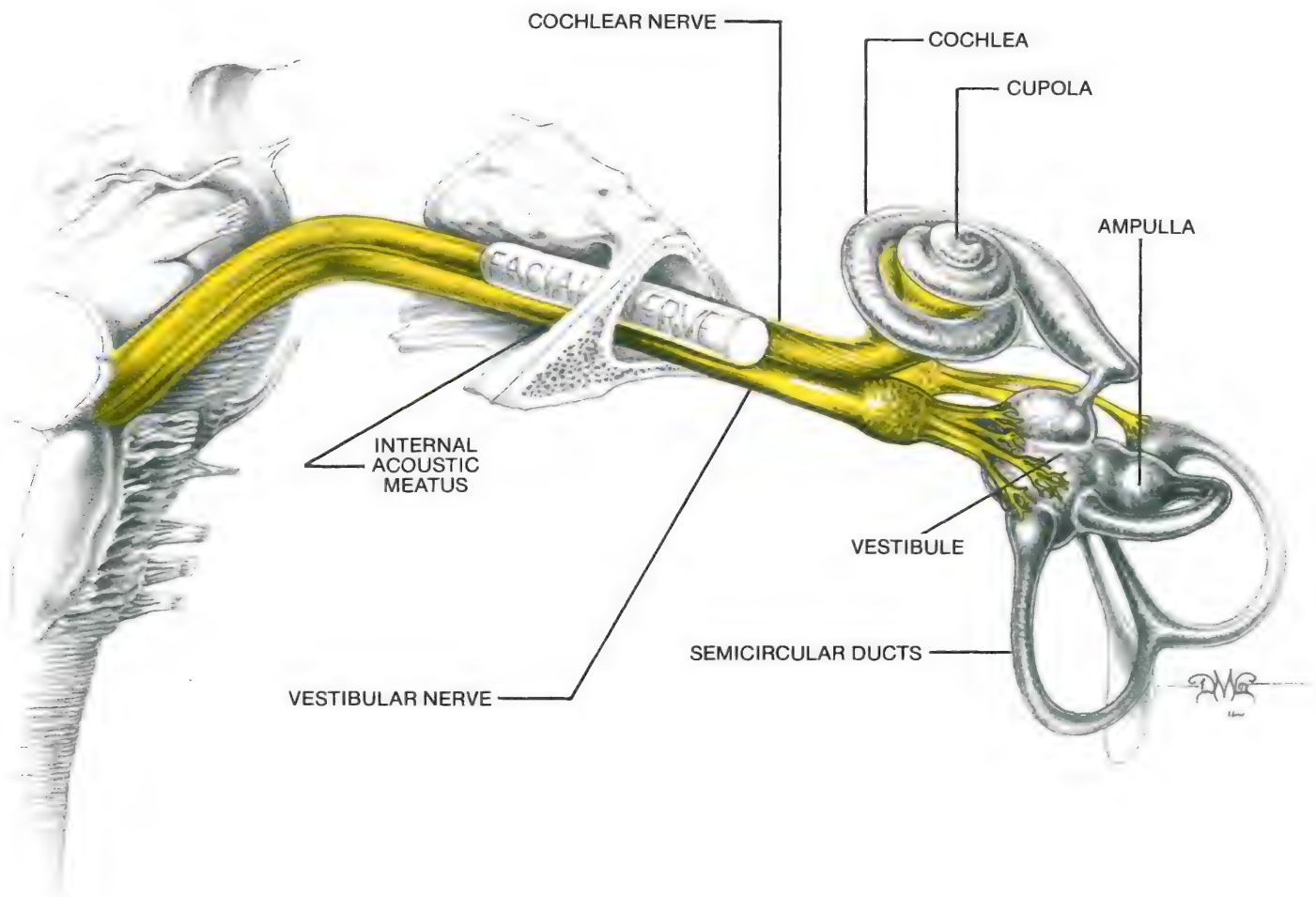
The acoustic nerve (vestibulocochlear nerve) consists of two separate sets of fibers which comprise the cochlear and vestibular nerves, each having significantly different functions. The superficial connection to the brain is between the pons and medulla oblongata. Both divisions of the nerve transmit afferent impulses of different types from the internal ear to the brain.

Cochlear Nerve

The cochlear nerve is for hearing. It arises peripherally in the spiral organ of Corti, exiting the temporal bone through the internal auditory canal, passing over the jugular tubercle and ending in the cochlear nuclei of the medulla. Each cochlear nucleus is connected with the cortex of both temporal lobes.

Vestibular Nerve

The vestibular nerve carries afferent impulses of equilibrium from the vestibular ganglion within the internal auditory meatus. It courses as a common trunk with the cochlear division from the internal auditory canal to the brain. Impulses from this nerve through the vestibulospinal tracts are involved with organization of the limbs and trunk in response to stimulation of the vestibular end organs. Eye movement in relation to head movement is organized through the medial longitudinal fasciculus, while muscular adjustment to control postural balance is organized through the cerebellum. It appears that applied kinesiology — in several of its techniques — is working with organization of the vestibular mechanism and other body proprioceptors for body organization.



5—11. Acoustic nerve (VIII)

POSSIBLE ENTRAPMENT

Trauma often involves the acoustic nerve and facial nerves in fractures, subdural hemorrhage, or inflammatory processes. Tumors in the cerebello-pontine angle may also involve both nerves. A functional entrapment may take place as a result of dural involvement, especially at the internal acoustic meatus.

Often the temporal bones are in counter-rotation,

disorienting the integration of the vestibular mechanisms.⁴¹ If this mechanical derangement causes signals of different head orientation to be sent from the two sides, it seems obvious confusion will result. This is especially important because of the integration which must take place between the labyrinthine, visual righting, and neck righting reflexes, as well as the other equilibrium reflexes of the body.

EXAMINATION

Examination of the cochlear and vestibular divisions involves two separate roots. Evaluation of either nerve is problematic; it is difficult to localize areas of dysfunction.

Cochlear division: Involvement of the cochlear division causes a disturbance in hearing which may be manifested as loss of hearing, tinnitus, or distortion of sounds. Clinical evaluation is limited primarily to determining whether consultation with a specialist in neuro-otology is necessary. Specialists' tests include pure-tone audiometry, speech audiometry, loudness balance test, tone decay, short increment sensitivity index, evoked auditory responses, and electrocochleography.¹⁷

Simple audiometers such as that illustrated in Figure 5—12 are available for screening evaluations of hearing in broad tone levels. Although the instrument cannot give the quantitative information available in the standard pure-tone audiometric test, it gives far better information than the evaluation of the subject's ability to hear a pocket watch ticking at various distances.



5—12. Speech frequency audiometer.

In the absence of an audiometer, a watch (preferably a pocket watch for its loudness) can be used to obtain a gross evaluation of hearing. The examiner determines the distance from the ear at which a normal person can hear the watch ticking. This information can be obtained by having several individuals with normal hearing listen for the ticking of the watch as it is moved closer to the ear. When evaluating a subject this way, each ear should be tested and compared with the other.

Rinne's test compares air conduction with bone conduction. Normally there is better ability to hear by air conduction than by bone conduction. The examiner places a vibrating tuning fork (C-256 vibrations per second) against the mastoid process of the individual, who is instructed to indicate when the sound ceases. When the individual can no longer hear the tuning fork, the examiner removes it from the mastoid process and places the vibrating end close to the external auditory meatus, without increasing the vibration of the fork. The patient should now be able to hear the vibration again, since air conduction should be better than bone conduction. An abnormal Rinne test is evidence of blockage of the external auditory canal or of middle ear defect. When the cochlear nerve is involved, there is diminished ability to hear the tuning fork by both air and bone conduction; however, there is still an increased ability to hear by air conduction.

Another test using the vibrating tuning fork is Weber's, where the vibrating tuning fork is held to the vertex of the head. Under normal conditions, the sound will be heard equally well in both ears. With middle ear disease or blockage of the external auditory canal, the sound will be louder on the side of hearing loss. In cochlear nerve disease, the sound will be heard better on the side opposite involvement; in other words, it is necessary to first determine the side of hearing loss by audiometric methods, and then compare the side of hearing loss with the side which better hears the vibration in the Weber test.



5—13. Rinne's test.



5—14. Weber's test.

Vestibular division: Evaluation of the vestibular branch begins with observation of the individual's orientation in space.

The Babinski-Weil test closely evaluates the patient's orientation in space. The patient walks forward approximately eight steps and then backward eight steps, with his eyes shut. If the trunk bends to one side when walking forward and to the other side when walking backward, disease of the internal ear is indicated — either osseous or membranous labyrinthitis. If the trunk bends to the same side when walking both forward and backward with the eyes closed, there is indication of probable ipsilateral cerebellar lesion.⁴³

Nystagmus is an involuntary rapid movement of the eyeball. Observation of spontaneous nystagmus can be a valuable diagnostic sign in revealing the location of a lesion responsible for dizziness. Nystagmus can be of the rotatory, vertical, horizontal, or oblique type. It has two components: (1) a slow movement in one of the directions, and (2) a fast return movement. The direction of nystagmus is considered to be that of rapid eye movement. Nystagmus is also graded as to frequency, amplitude, duration, multidirection, and dissociation of the eyes. These factors are all taken into consideration to determine the possible area of lesion.²¹

The usual procedure for evaluating vestibular function is to irrigate the external auditory canal with cold water. The patient's head is elevated 30° from the horizontal, which places the lateral semicircular canal in a vertical plane. The external auditory canal is irrigated with 20 cc of 30° C water over a period of twenty seconds. During the procedure, the patient's eyes should be open, looking straight ahead so that immediately after the procedure the examiner can observe for nystagmus. The direction, frequency, and duration are recorded. The duration is usually approximately two minutes. This is a minimal test, but it will develop nystagmus in most normal individuals. If nystagmus fails to develop from this irrigation, the test is repeated with 30 cc of water over a thirty-second period of time. Failure to develop nystagmus is strong evidence of hypofunction of the labyrinth. Differentiation between a central or vestibular lesion requires more complex caloric testing, which usually requires referral to a specialist.²¹

There are several more sophisticated tests which include electronystagmography, otokinetic stimulation, rotational tests, cold air test when water irrigation is contraindicated (as in the case of middle ear infection), optokinetic drum test, and galvanic test. (For a more thorough discussion of evaluating the dizzy patient, see Volume V.)

IX — Glossopharyngeal Nerve

ANATOMY AND DISTRIBUTION

The glossopharyngeal is a mixed nerve with motor fibers to muscle and viscera; likewise, the sensory fibers are both visceral and somatic. It supplies motor fibers to the stylopharyngeus muscle, and secretomotor fibers to the parotid and small glands in the mucous membrane of the posterior part of the tongue and neighboring pharynx. Afferent fibers come from the pharynx and posterior portion of the tongue, supplying both taste and general sensation. It also supplies the tonsils, carotid sinus, and the styloglossus muscle with sensory fibers. A small portion of skin anterior to the tragus and some of the anterior wall of the external auditory meatus

are also supplied with afferent fibers.

The glossopharyngeal's superficial origin is from the superior part of the medulla oblongata near the vagus nerve. The nerve courses laterally toward the jugular foramen, with its own sheath of dura mater in contact with the petrous portion of the temporal bone. As it traverses the jugular foramen, there is a small superior ganglion in the upper part of the groove and an inferior ganglion in the lower portion. In these ganglia are cell bodies for the sensory fibers of the nerve. It courses inferiorly to its distribution through several branches.

POSSIBLE ENTRAPMENT

A functional entrapment at the jugular foramen can interfere with the glossopharyngeal nerve. Generally when there is entrapment at this area, the vagus (X) and/or the spinal accessory nerves (XI) will also be involved. Whether the entrapment is nerve damaging (such as a fracture may be) or a functional problem (such as a cranial fault) the symptoms of the

vagus, and possibly the spinal accessory, will overshadow those of the glossopharyngeal. Neurologic evaluation may pick up involvement of this nerve when symptoms give no indication.

Indication of glossopharyngeal involvement can be from intramedullary or extramedullary lesions, neoplasms, or inflammatory reactions.

EXAMINATION

Examination of the glossopharyngeal nerve is more limited than the examination of most of the other cranial nerves. The most common test used for its evaluation is the gag reflex test, which involves stimulating the posterior pharyngeal wall with a tongue depressor. The usual response is a contraction of the pharyngeal muscles, generally resulting in a gag, though this is not necessary for a normal response. It is pointed out, however, that after intercranial sectioning of the glossopharyngeal nerve a normal gag reflex may still be present, indicating that the posterior pharyngeal wall is also supplied by the vagus nerve (X), making this an unreliable test for glossopharyngeal nerve function.⁴⁴

Evaluating taste function on the posterior one-third of the tongue is problematic. It is best done by anodal galvanic stimulation, which is perceived as acidic or metallic taste.^{33, 34}

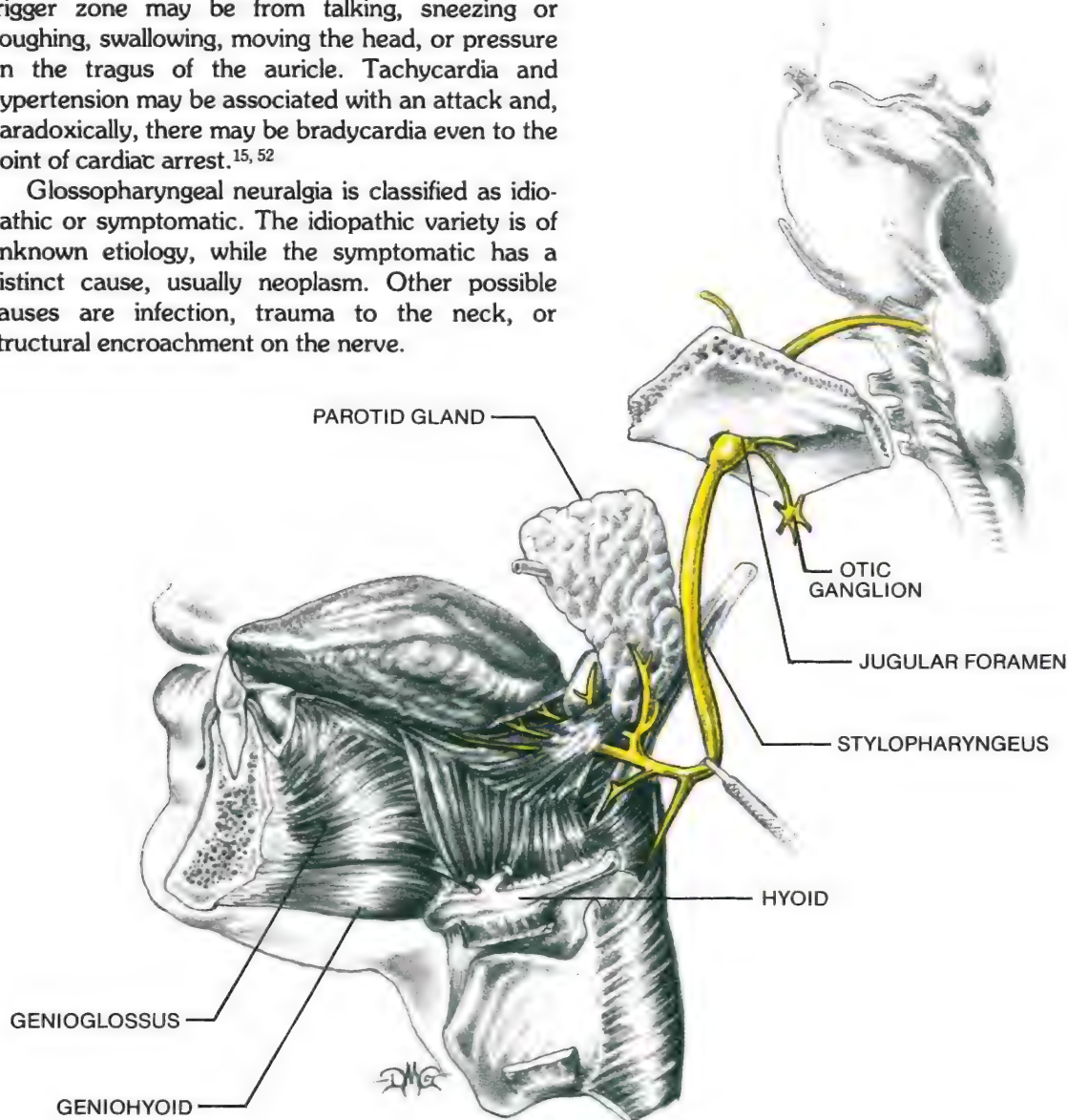
Normally, pressure on the carotid sinus causes a slowing of the heart and is listed as one of the examination procedures for the glossopharyngeal nerve. It is recommended that this not be used as a routine evaluation because it has potential hazards; fatal ventricular fibrillation may develop.¹ Avoiding this procedure is particularly important in individuals with vascular disease who are taking medication for it.⁹ The patient taking digitalis is more susceptible to ventricular fibrillation from carotid sinus stimulation.⁵¹ Specifically, bilateral carotid sinus stimulation should never be used, and unilateral stimulation should not exceed five seconds in duration at any time. Electrocardiographic monitoring should be used for all patients over forty; when ECG is not used, auscultatory monitoring should always be performed. Avoid carotid sinus pressure when the blood pressure is initially below 100 mm Hg systolic.

Elderly patients and those with known vascular disease should not be examined or treated in this manner.²⁷

Glossopharyngeal neuralgia can be difficult to differentiate from trigeminal neuralgia affecting the mandibular nerve. As with trigeminal neuralgia, the pain is generally unilateral and paroxysmal with a sharp, stabbing quality. The pain is usually located at the root of the tongue, possibly radiating toward the angle of the jaw and the ear. The trigger zone which tends to initiate a paroxysm of pain may be in any or all of the following areas: tonsil, posterior pharynx, and tragus of the auricle. Adequate stimulation to the trigger zone may be from talking, sneezing or coughing, swallowing, moving the head, or pressure on the tragus of the auricle. Tachycardia and hypertension may be associated with an attack and, paradoxically, there may be bradycardia even to the point of cardiac arrest.^{15, 52}

Glossopharyngeal neuralgia is classified as idiopathic or symptomatic. The idiopathic variety is of unknown etiology, while the symptomatic has a distinct cause, usually neoplasm. Other possible causes are infection, trauma to the neck, or structural encroachment on the nerve.

The improvement in hypertension seen as the result of correcting cranial faults may be related to the glossopharyngeal nerve. This is particularly true if the reduced blood pressure observed from cranial correction is of only a transient nature. The carotid sinus adapts to long-term blood pressure variations. These baroreceptors adapt themselves in one to three days to whatever pressure level they may be exposed.²⁸ Acute bilateral intercranial sectioning of the nerves has been shown to cause immediate but transient hypertension.⁷⁴



5—15. Glossopharyngeal nerve (IX).

X — Vagus Nerve

ANATOMY AND DISTRIBUTION

The vagus nerve has a large distribution. It is a mixed nerve — sensory and motor — with somatic and visceral fibers in both aspects. Its superficial origin is from the medulla oblongata between the glossopharyngeal and accessory nerves. It courses over the jugular tubercle to the jugular foramen. Passing through the jugular foramen, it is contained in the dural sheath it shares with the spinal accessory nerve. Within the jugular foramen, the vagus demonstrates the superior ganglion; it presents another ganglion — the inferior — shortly after it exits the foramen. The nerve is joined by the cranial portion of the spinal accessory nerve and has a wide distribution, with motor fibers to the pharynx, larynx, base of the tongue, and through autonomic ganglia it innervates structures in the thorax and abdomen. There are sensory fibers from the ear, pharynx, larynx, and the thoracic and abdominal viscera.

The auricular branch is from the superior ganglion, which supplies somatic afferent fibers to the external auditory meatus and the external ear. The inferior ganglion (nodose ganglion) gives off the pharyngeal branches which join the pharyngeal plexus to supply the mucous membrane of the pharynx and the muscles of the soft palate, except the tensor veli palatini.

The superior laryngeal nerve is from the inferior

ganglion. It communicates with the superior cervical and sympathetic ganglia and may contribute — with the pharyngeal plexus — to supply the carotid body. The carotid body has chemoreceptors monitoring the oxygen tension of the blood and baroreceptors monitoring the blood pressure. The superior laryngeal nerve branches to supply muscles and mucous membranes of the pharynx, and it also branches to the epiglottis, base of the tongue, and part of the larynx.

The cardiac plexus is formed from the cardiac branches, sympathetic postganglionic fibers from the three cervical sympathetic ganglia, and nerves from the other side. The vagi in the thorax have a considerable interchange; they supply branches to the bronchi, esophagus, and lungs.

There are both visceral efferent and afferent fibers in the abdomen. The gastric branches spread over the fundus and body of the stomach, entering the stomach wall to supply the myenteric and submucous plexuses.

The hepatic branches supply the liver, gall bladder and bile ducts, pancreas, pylorus, and part of the duodenum. Celiac branches join the celiac plexus from which vagus fibers supply the pancreas, kidney, spleen, and small and large intestines.

POSSIBLE ENTRAPMENT

The vagus nerve, like the glossopharyngeal, can encounter entrapment at the jugular foramen from trauma, and it also appears to develop a functional-type entrapment from cranial dysfunction. Generally there will be an involvement of the spinal accessory nerve also, as the two nerves travel in the same dural sheath. Further, there may be entrapment outside the cranium of a functional, structural nature as the nerve courses through the complex of muscles, fascia, and other structures. Structural correction of

cranial faults, subluxations, neck involvements, and general body imbalance, appears to have a major influence on correcting functional disturbances of the vagus nerve.

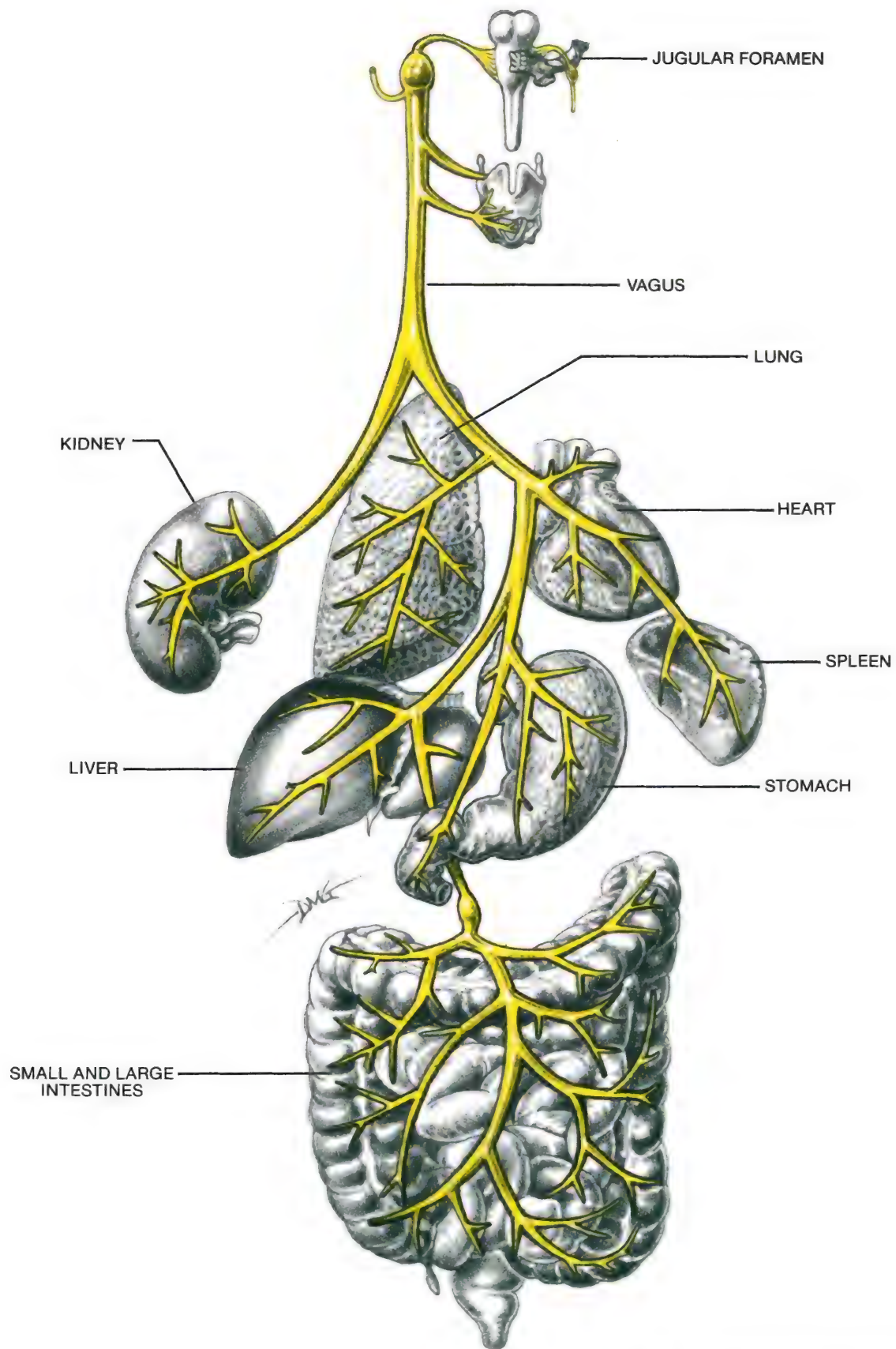
Lesions that are intermedullary or extramedullary and outside the cranium in the form of neoplasms, inflammatory processes, toxic agents or metabolic disorders, must be considered when there is evidence of vagal dysfunction.

EXAMINATION

Despite its wide distribution, vagal examination on an objective basis is problematic. There are a minimal number of tests to perform. With few signs available, symptoms are often the leading evidence of

vagal disturbance. Generally, the symptoms can relate to cardiovascular, laryngeal, digestive disturbances, and dysphagia.

Bradycardia can develop with irritative lesions,



5—16. Schema of Vagus nerve (X).

Cranial Nerves

while paralysis of the vagus will cause tachycardia. A test which has been used to evaluate this action is pressure on the carotid sinus or on the eyeball to increase vagal activity and establish bradycardia. The value of these tests as a routine measure should be questioned. In susceptible individuals with cardiac disturbance they should not be done. Severe reactions to the test — fainting or even death — have been observed.^{1, 9, 27, 45, 51} (See page 128.)

A test using atropine to paralyze the vagus to determine change in heartbeat is a medical approach of evaluation.¹⁵ To determine if complete interruption of supply has been accomplished following vagotomy, insulin is injected to create hypoglycemia which causes hydrochloric acid secretion when there are intact fibers.³⁰ The vagus stimulation may be from absolute levels of glucose or from the rate of fall; which is the case is not known. In any event, when a functional blood sugar or vagal disturbance is present the physician is alerted to the possibility of an interrelationship.

The larynx may be involved unilaterally or bilaterally. It may manifest dysfunction as a voice that is deep and hoarse. If the condition is unilateral, the symptom depends on the ability of the functioning opposite cord to compensate if the paralyzed cord is not in the median position. When there is bilateral paralysis, the vocal cords are in the “cadaveric position.” This is the position found in tranquil respiration. Normally they are closed during phonation and widely opened in deep inspiratory efforts. When there is paralysis and the cords are in the

cadaveric position, there is usually also palatal and pharyngeal paralysis with the nerve lesion central to the inferior ganglion.

Dysphagia can be either severe or observed periodically. Muscular action to elevate the palate to close the nasopharynx must take place as the epiglottis is raised, closing the opening to the larynx. Failure of this activity at the appropriate time in swallowing can allow fluids to enter the larynx, causing a spasmodic cough with regurgitation of fluids into the nasopharynx. This happens periodically with functional disturbance; it may be constant when there is a nerve lesion. There may be salivary disturbances, with temporary hypersecretion with irritative lesions and hyposcretion with paralysis. Snoring, alterations in phonation, and palatal paralysis causing nasal-sounding speech are all indicative of possible dysfunction.

Digestive dysfunction develops with vagal involvement; however, it is very difficult to objectively track down as being induced by vagal dysfunction. A unique factor that can occur after vagus nerve damage is abnormal regeneration of the nerve. This is when the regenerated fibers cross-connect to innervate sweat glands, producing a condition called gustatory sweating. In this condition, the patient begins to sweat as a result of any condition that would ordinarily cause salivary or gastric secretions. Eating — or even just the sight of food — can induce impulses to cause the sweating. This condition can develop as a result of thoracotomy, or surgery at the root of the neck.

XI — Spinal Accessory Nerve

ANATOMY AND DISTRIBUTION

The spinal accessory nerve is largely a motor nerve which has an unusual arrangement of its two parts, the cranial and spinal. Some texts refer to the cranial part as the inferior portion of the vagus nerve and call it the accessory nerve. Other texts refer to cranial nerve XI as the accessory nerve, including both the cranial and the spinal portions of what is termed here the spinal accessory nerve. This leads to confusion which must be kept in mind when referring to various texts.

Cranial Part

The superficial origin of the cranial part is from the medulla oblongata, close to the vagus. It courses laterally to the jugular foramen, over the jugular

tubercle. It interchanges some fibers with the spinal part, or becomes united with it for a short distance. It also has a few fibers interchanging with the vagus nerve.

After traversing the jugular foramen, the cranial part separates from the spinal portion and joins the vagus at its inferior ganglion. The fibers of the spinal accessory cranial root probably distribute to the pharynx, larynx, soft palate, esophagus, and cardiac nerves.

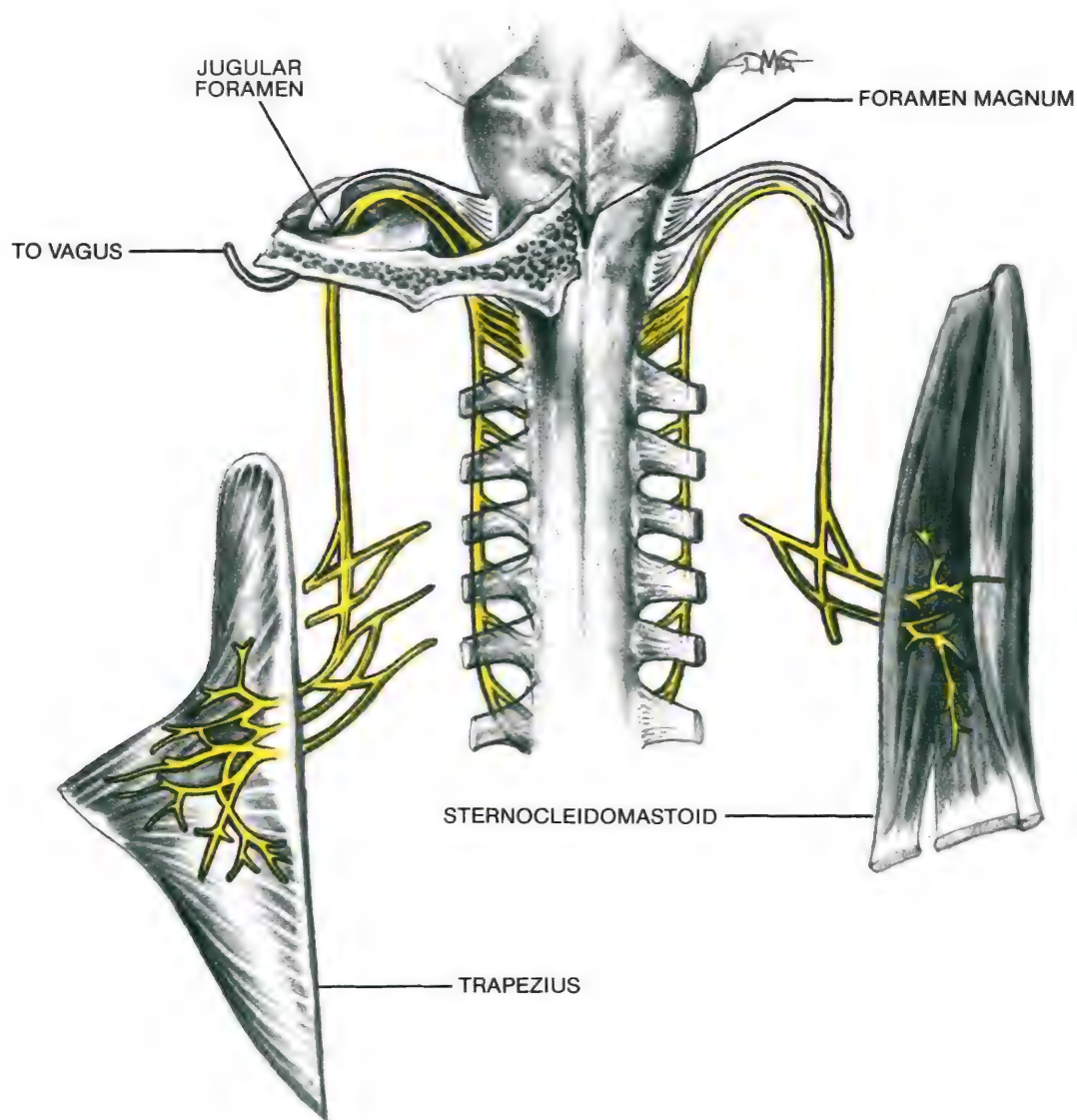
Spinal Part

The spinal part originates at the junction of the spinal cord with the medulla oblongata in the lateral part of the anterior gray column, extending down six

cervical segments.⁴⁸ Passing through the lateral white column of the spinal cord, they emerge on its surface midway between the ventral and dorsal nerve roots of the upper cervical nerves and unite to form a trunk. The nerve ascends through the foramen magnum into the cranial cavity. It traverses the occipital bone to the jugular foramen, where it passes in the same sheath of dura mater as the vagus nerve. In the jugular foramen, it may receive filaments from — or join — the cranial root for a short distance. Outside the jugular foramen, it is separate from the cranial root, coursing posterolaterally to descend to the upper part of the sternocleidomastoid muscle, joining with branches of the 2nd and possibly 3rd

cervical nerve(s) to supply the muscle. From here, the spinal part of the spinal accessory nerve courses across the posterior triangle of the neck to the trapezius. It communicates with the 2nd, 3rd, and 4th cervical nerves, forming a plexus which innervates the muscle.

The sternocleidomastoid and trapezius muscles receive motor innervation from the spinal accessory nerve, while they receive their sensory innervation from the cervical nerves. Although this seems to be the general consensus, there is controversy over this arrangement. There appears to be some afferent supply by cranial XI.^{48, 77}



5—17. Spinal accessory nerve (XI).

POSSIBLE ENTRAPMENT

The spinal accessory nerve can have the same entrapment described for the vagus, within the cranium and at the jugular foramen. In applied kinesiology, evaluation of this nerve is easily done by manual muscle testing of the sternocleidomastoid and upper trapezius muscles. Entrapment as a result of cranial faults appears to be a relatively common occurrence. Weakness of the sternocleidomastoid or upper trapezius muscle is often observed by manual muscle testing, and is most often immediately returned to normal strength after cranial fault correction.

Neoplasms, infection, and subdural hematoma — as well as other pathologic and traumatic conditions — can cause entrapment of the spinal accessory nerve, as with other cranial nerves. Trauma to the neck region and structural distortion of a functional nature can cause entrapment of the peripheral nerve after it leaves the jugular foramen. There may be trauma or entrapment of the spinal division at the foramen magnum.

The unusual innervation of the sternocleidomastoid and upper trapezius is of interest, especially to those working with the cranial primary respiratory mechanism. The efferent supply to these muscles is

by way of the spinal accessory nerve. Although the cells of origin are in the posterolateral part of the anterior horn of the spinal cord, the nerve travels through the cranium to exit at the jugular foramen, thus being subject to the functional type of nerve entrapment associated with cranial faults and also upper cervical subluxations. The afferent supply to the sternocleidomastoid and upper trapezius is primarily from the anterior rami of C2, 3, and 4. There is some afferent supply from the spinal accessory with the cell bodies within the intercranial portion of the nerve trunk.^{48, 76} These fibers are probably muscle afferents.⁷⁷ This unusual innervation appears to be important in leveling the head on the body in certain types of conditions observed in applied kinesiology. If there is entrapment with subsequent dysfunction of the efferent supply to the muscles, with no involvement of the sensory supply, there will be confusion of body function. The same is true if the interference is on the afferent supply from a cervical subluxation and efferent supply is normal. Goodheart hypothesizes a reason for the dual innervation of these muscles as being part of the gait mechanism (see Chapter 17).

EXAMINATION

Examination of the spinal accessory nerve involves testing the sternocleidomastoid and upper trapezius muscles. Standard procedures of manual muscle testing used in applied kinesiology are used to evaluate the nerve (see Volume I for muscle testing procedures). Generally when there is entrapment of the spinal accessory nerve at the jugular foramen, there will be weakness of both the sternocleidomastoid and the upper trapezius. This is not always the case, however, since it appears that individual fibers of the nerve can be affected; sometimes the upper trapezius also receives its efferent supply entirely by spinal innervation. There is considerable anatomical variance of the distribution of the spinal accessory nerve.¹³ The use of AK techniques of therapy localization and challenge can help determine which area is involved and applicable for conservative treatment.

Eisen and Bertrand¹⁹ describe four cases of unilateral accessory nerve palsy of an idiopathic nature. The involvements were confirmed by electro-

physiologic measures. Shoulder position and activity were markedly affected. They describe the condition as relatively rare. AK clinical experience shows the involvement not to be uncommon as it often appears to be the cause of shoulder problems, especially of the “frozen shoulder” variety. It does appear that their four cases were more progressed than those routinely seen in an AK practice. This seems to point out the value of examination to find functional disturbances.

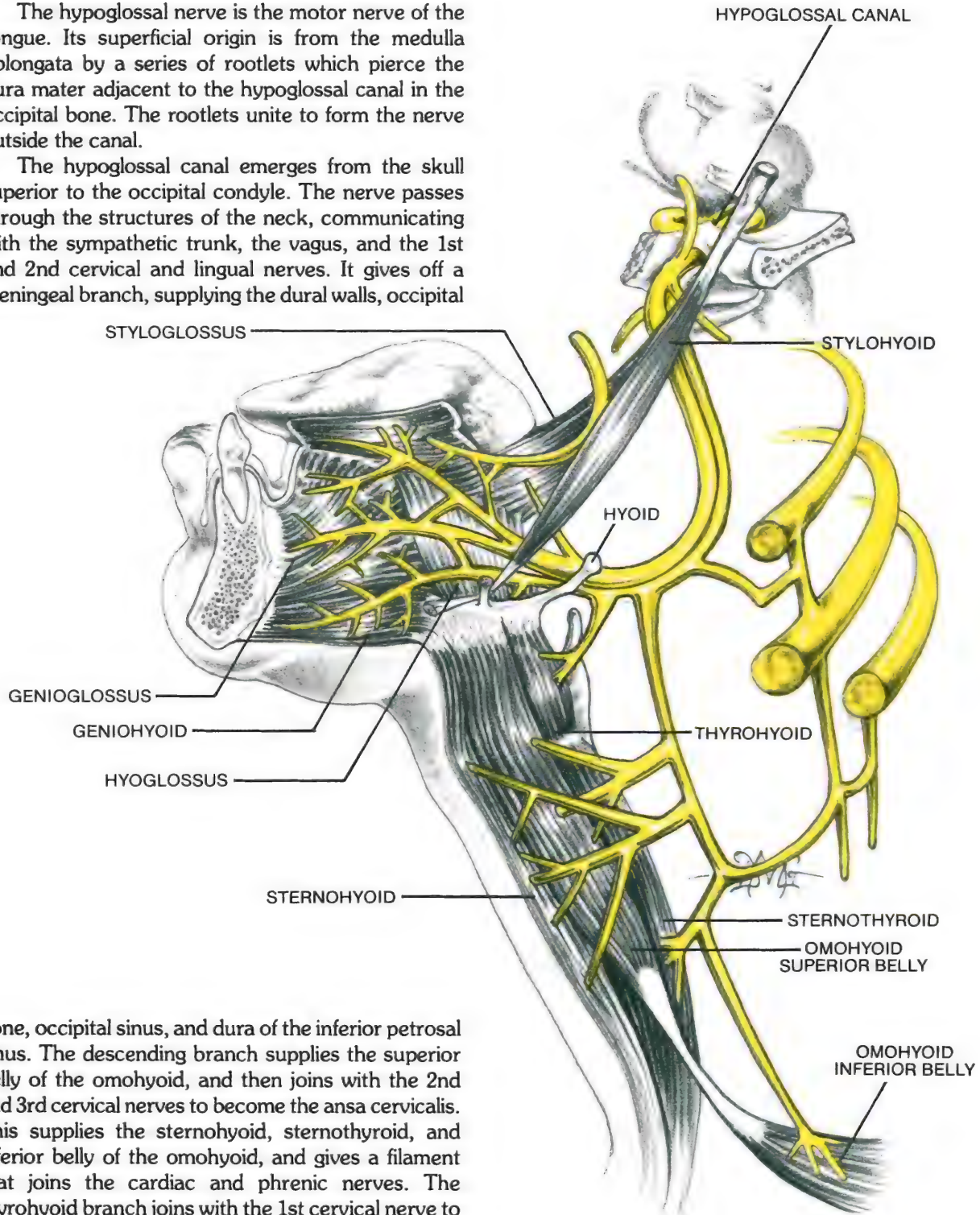
Standard postural evaluation as used in applied kinesiology indicates involvement of these muscles on a functional or pathological basis. Differentiating functional from pathological (neoplasm, inflammatory processes, subdural hemorrhage, etc.) is usually relatively easy, as functional conditions respond immediately in nearly all cases; the pathological involvement does not respond to a therapeutic trial, and there is usually muscle atrophy if the condition has any chronicity.

XII — Hypoglossal Nerve

ANATOMY AND DISTRIBUTION

The hypoglossal nerve is the motor nerve of the tongue. Its superficial origin is from the medulla oblongata by a series of rootlets which pierce the dura mater adjacent to the hypoglossal canal in the occipital bone. The rootlets unite to form the nerve outside the canal.

The hypoglossal canal emerges from the skull superior to the occipital condyle. The nerve passes through the structures of the neck, communicating with the sympathetic trunk, the vagus, and the 1st and 2nd cervical and lingual nerves. It gives off a meningeal branch, supplying the dural walls, occipital



bone, occipital sinus, and dura of the inferior petrosal sinus. The descending branch supplies the superior belly of the omohyoid, and then joins with the 2nd and 3rd cervical nerves to become the ansa cervicalis. This supplies the sternohyoid, sternothyroid, and inferior belly of the omohyoid, and gives a filament that joins the cardiac and phrenic nerves. The thyrohyoid branch joins with the 1st cervical nerve to supply the thyrohyoid muscle. There are muscular branches to supply the styloglossus, hyoglossus, geniohyoid, and geniohyoid.

5—18. Hypoglossal nerve (XII).

POSSIBLE ENTRAPMENT

The hypoglossal nerve probably develops functional entrapment from cranial faults as it pierces the dura mater and enters the hypoglossal canal. This should be differentiated from neoplasm, trauma

directly affecting the nerve, and conditions which may involve the anterior horn cells in the spinal cord, such as motor neuron disease and poliomyelitis.

EXAMINATION

Standard examination deals primarily with observing the tongue's function and testing its strength. The patient is asked to extend his tongue while the examiner watches for lateral deviation. The tongue will deviate to the side of a unilateral lesion as a result of the muscle's lack of function on that side. The strength of the tongue can be observed by having the patient protrude it into the cheek, causing a bulge which the examiner palpates for strength. Another method is to have the patient protrude his tongue laterally, and the examiner attempts to push it toward the midline with a tongue depressor. In chronic conditions, atrophy may be observed ipsilateral to the side of lesion. It may be necessary for the examiner to palpate the tongue to observe unilateral atrophy. Having the patient extend his tongue in an upward and downward position, as if to lick his upper and lower lips, may provide information not observed with other testing procedures.

If there is a bilateral lesion, the entire tongue will be paralyzed. In total paralysis, it is necessary for the

patient to push a food bolus to the pharynx with his finger before he can swallow it. In an upper motor neuron lesion, the ability to wiggle the tongue from side to side will be very slow.

Although there are afferent fibers in the hypoglossal nerve, the tongue will maintain its sensation and taste ability in the presence of a lesion because of nerve supply from the chorda tympani and other facial nerves.

A burning sensation of the tongue is called glossodynia. The burning sensation may extend throughout the mucus of the mouth. Generally there is deep fissuring of the tongue and the condition is due to a vitamin B complex deficiency.²⁰ This condition is more often seen in middle-aged and elderly patients, and more frequently in females than in males. It responds rapidly to vitamin B complex support, but often requires a very natural form of low potency B complex with the patient chewing the substance.

Differentiation of the Etiology of Cranial Nerve Dysfunction

At this time, most doctors working with cranial primary respiratory dysfunction are those who deal with functional disturbances. A functional disturbance refers here to one where disease — such as neoplasms, aneurysms, infection, and toxic disturbance — has not affected the nerve or its central nucleus. It also excludes nerve dysfunction as a result of trauma which may directly tear nerve tissue or cause subdural hemorrhages, aneurysms, etc., possibly resulting in an encroachment on nerve tissue. It is therefore important to emphasize the necessity of differential diagnosis to indicate when a condition may be corrected by improving the primary respiratory mechanism's function, and when it is necessary to refer the patient to the appropriate practitioner for further evaluation and possible treatment or surgery. This may include angiograms, CAT scan, electroencephalography, nerve conduction studies, and other specialized procedures. Many

specialty procedures have been mentioned regarding examination of the various cranial nerves. This is to acquaint the general practitioner with the available diagnostic methods which may be outside his area of practice. The clinician knowledgeable in cranial nerve evaluation can usually readily make the necessary differentiation regarding the necessity for referral.

When there is evidence of several cranial nerve involvements, the areas of possible functional entrapment are considered to determine if the combined dysfunction could be functional. In most cases of pathological involvement, the pattern will be such that it is improbable that cranial faults could be the etiology. For example, there is generally no evidence of bilateral entrapment of cranial nerves IX, X and XI at the jugular foramen as a result of cranial faults. Bilateral dysfunction would seem to indicate a more central lesion.

When there is evidence of unilateral entrapment of cranial nerves IX, X, and XI, the clinician working with the cranial primary respiratory mechanism should evaluate for functional cranial faults. If any are found, correction will nearly always immediately improve the function of the nerves, reducing the positive clinical evidence of involvement. There are occasions when pathologic dysfunction is concurrent with cranial faults. In this case, cranial correction often seems to improve the condition; however, there will usually be a rapid return of dysfunction or a limited amount of improvement.

Conditions resulting from recent trauma especially should be examined closely to rule out the possibility of direct trauma to nerve tissue or conditions such as subdural hematoma, intracerebral hemorrhage, etc.

One of the values that applied kinesiology has

added to the treatment of cranial faults is the ability to objectively determine when improvement has been obtained. This has not only helped to improve therapeutic efforts; it is also of value in differential diagnosis. Clinical experience indicates that the examination, application of treatment, and evidence of improvement of function should follow a predictable pattern. Thorough knowledge of the AK approach allows the physician to determine whether the patient's condition is following the pattern expected. If the cranial examination — including cranial nerve evaluation — shows a confusing pattern compared to that of usual functional disturbances, or if treatment does not obtain the usual improvement of function, or if — after thorough evaluation and correction — improvement is not maintained, then consultation with a neurologist should be considered.

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Chapter 6

Examination and Treatment of Cranial Faults

Introduction

Applied kinesiology has named and treats various types of cranial faults, using specific examination procedures and corrective approaches. These faults provide a good, basic working approach to the cranium for study purposes. Although these faults are listed as specific entities, it must be observed that there is a continuum among various types of faults. The cranium functions as an integrated whole, with the reciprocal tension membrane an integral part. An individual may exhibit many cranial faults when first examined. These can sometimes be corrected with one therapeutic effort into the system because of the effects of the reciprocal tension membrane and the gear-like mechanisms of the sutures. When a corrective force is applied to a key area in the cranial mechanism, many cranial faults other than the one specifically being treated will often be eliminated. An understanding of the cranial mechanism, along with accurate evaluation, aids the physician in using minimal therapeutic effort to gain maximum correction.

Emphasis has been placed on the way the various bones move in the cranial primary respiratory

scheme. It is important to learn these motions so the knowledge can be applied to examination procedures which make use of the continuum of cranial faults. After the physician has learned the basic faults and their corrections, he will develop an ability to evaluate the skull for its individual characteristics.

Early in the development of applied kinesiology application to the cranial primary respiratory mechanism, little effort was made to evaluate the individuality of each skull. This effort will be emphasized throughout the examination and correction procedures because of its importance in efficient, maximum correction and the elimination of iatrogenic problems which can develop as a result of cranial therapeutics.

This chapter first explains the correlation between diaphragmatic respiration or breathing and the cranial primary respiratory mechanism. There is then a discussion of the body language presented by cranial faults, an introduction of various examinations used for the cranium, and, finally, the procedures used in applied kinesiology to correct the cranial primary respiratory mechanism.

Diaphragmatic and Cranial Respiratory Correlation

The cranial-sacral primary respiratory mechanism has been previously described as an autonomous cycling which takes place independently of other body cycles. There is a correlation between thoracic respiration and the cranial-sacral motion. Deep respiration of the thorax influences motion of the cranium and pelvis. Although this influence is always present, relaxed breathing and primary respiration do not always parallel.

Most of the cranial faults listed in applied kinesiology have a specific thoracic respiratory correlation which can be observed in several ways. A muscle which is weak because of a cranial fault will strengthen when the patient holds a phase of thoracic respiration which helps correct the fault. When the cranial fault has been corrected, the muscle will be strong on any phase of respiration.

This respiratory assist appears to place the skull

Examination and Treatment of Cranial Faults

in an improved position to avoid interference with normal nerve function. Holding a deep thoracic inspiration places the skull in its maximum sphenobasilar flexion. If the skull is in abnormal extension during quiet respiration, thus causing interference with normal nerve function, the correction needed is to place the skull in its neutral position and eliminate excessive sphenobasilar extension. This can be done by using the patient's thoracic respiration as a test to determine what phase causes a muscle which was weak as a result of a cranial fault to strengthen. Because a deep phase of thoracic inspiration places the skull in greater sphenobasilar flexion, nerve function is improved while the thoracic inspiration is held; thus the muscle weak because of the cranial fault becomes strong. This is illustrated in the accompanying schematic drawings, A — D. In A and B the solid lines indicate the normal relaxed breathing position for the sphenoid and occipital bones. The gray lines of A indicate the full range of sphenobasilar flexion, while the gray lines in B illustrate extension. In C and D, the relaxed position of the sphenoid and occipital bones is in excessive extension. This represents a cranial fault. Inspiration improves the position of the sphenoid and occipital bones, but they do not go to the full range they would normally move. In this case, a muscle which tests weak as a result of the cranial fault will have improved function when a thoracic inspiration is held and the muscle is tested. Expiration places the sphenoid and occipital bones in greater extension, or into greater lesion.

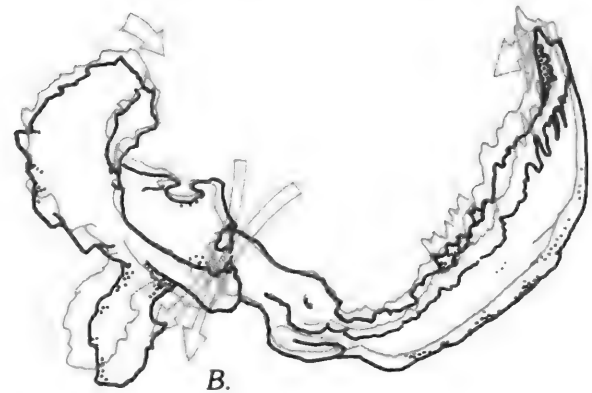
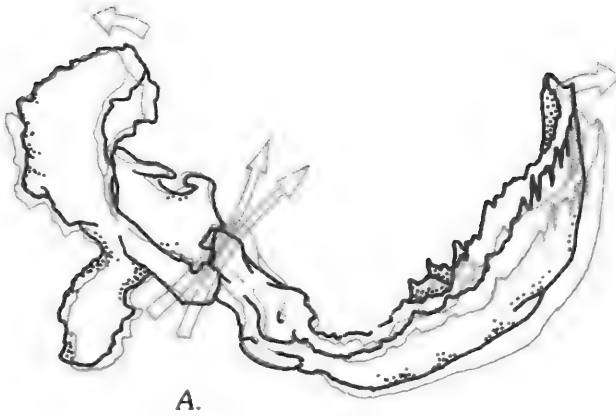
Another method of evaluating the respiratory correlation of cranial faults is to observe the change of a previously strong muscle (indicator muscle) on different phases of respiration. In D, the sphenoid and occipital bones are in excessive extension in the relaxed breathing position. Full expiration causes the bones to be placed further out of their normal position, into greater lesion. This appears to cause generalized confusion in the neuronal pools of the body, since almost any indicator muscle will test weak on manual muscle testing while this phase of respiration is held. In other words, the expiration causes an increased cranial fault, further interfering with normal nerve function. The involvement may be a subclinical fault, which simply means that under usual testing procedures the body appears to be functioning normally on a primary respiratory basis; however, when subjected to the complete range of respiratory motion, faults appear.

In both of the above cases where a weak associated muscle becomes strong on inspiration and a previously strong muscle weakens on full expiration, there is indication that the cranial fault is

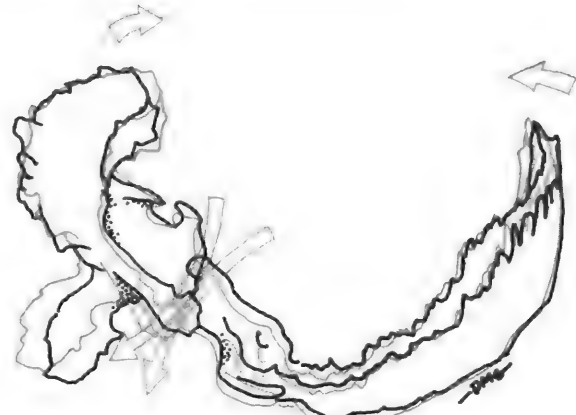
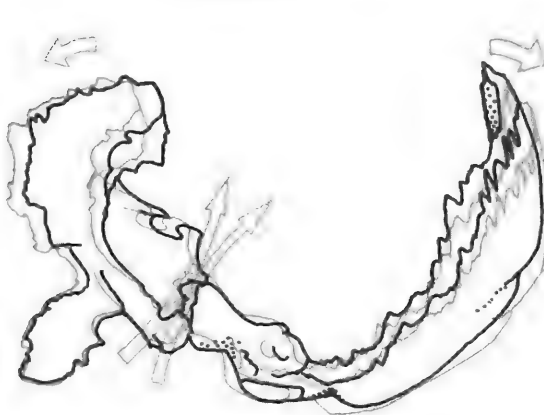
of increased sphenobasilar extension. This means that correction requires decreasing the sphenobasilar extension, which is done on a rebound basis. The respiration correlation just explained is a test only; it does not make any corrections (corrections will be discussed later). First it is important to determine the type of involvement present.

Another aspect of the working hypothesis is that there may be a locking within the cranium, limiting normal range of motion. In this case, the sphenoid and occipital bones may not be able to move in normal flexion on a deep phase of thoracic inspiration. Sensory information of motion failure may be transferred to the neuronal pools, creating confusion which interferes with normal function and is evidenced by a previously strong indicator muscle weakening as long as the phase of respiration is held. Information of motion failure may be transmitted from the afferent nerve endings in the sutures or dura mater.

An experiment can be designed to exemplify the change of muscle strength as a result of the skull's inability to go through its full range of motion. This experiment should be performed on an individual who does not appear to have any cranial faults. This can usually be determined by the individual's muscles being strong on all phases of respiration. To do the experiment, the examiner restricts normal skull motion by applying light digital pressure on a bone against the direction in which that bone usually moves with a specific phase of thoracic respiration. After the respiration the subject resumes normal breathing, and the examiner releases his pressure. There will be a weakening of a previously strong indicator muscle that will last for many seconds to a few minutes, until the skull resumes normal function on its own. For example, on inspiration the squama of the temporal bone moves laterally. If a gentle medial pressure is held on the superior squama of both temporal bones while the individual inspires, there will be an immediate and dramatic weakening of a previously strong indicator muscle, indicating the inability of the temporal bone squama to move laterally as it desires with that phase of respiration. The weakening will probably even be exaggerated if the tip of the mastoid process is held from moving posteriorly and medially as it normally does on deep thoracic inspiration. In doing this experiment, it is important that the bone's normal motion be resisted gently, and that as soon as the subject has completed the deep thoracic inspiration the bones are released by the examiner. This prevents creating a fault; continuing to hold the bones during the muscle test changes the concept of the experiment. Remember, this experiment must be done on an individual free of



Black lines represent the normal neutral position of the sphenoid and occiput bones. The gray lines in A represent exaggerated position on inspiration and in B on expiration.



C. Gray represents inspiration assist which improves the bones' position. This strengthens a muscle that is weak as a result of this fault.

D. Gray represents expiration making fault worse; strong indicator muscle weakens.

6—1. Schematic representation of motion on phases of respiration.

cranial dysfunction. On an individual with cranial faults the experiment may or may not work, depending on the type of cranial faults present.

The experiment just discussed has been used to evaluate the apparent motions of the cranial bones during different phases of respiration. The motions of certain bones have been controversial among various providers of cranial therapeutics. Where possible, the motions of the bones as outlined in this text have been confirmed by this method of evaluation. In nearly all instances, the bones appear to move in the direction originally outlined by Sutherland¹⁵ and further described by Magoun.¹¹

After discussion of the anatomy and motion of the individual cranial bones, there is a summary of total skull motion. The use of the experiment described above is again emphasized there. Its use by the individual practitioner to evaluate the motion of various skulls is a valuable learning experience

which will help emphasize skull individuality. Applied kinesiology methods of examination take this into consideration (see Chapter 3, page 87).

When correction is discussed, there will be a correlation of the different types of respiration which are used to aid correction of specific cranial faults. At this point it is important to understand the hypothesis of *why* muscle strength changes with different phases of thoracic respiration.

To summarize the hypothesis, a weak muscle associated with the cranial fault becomes strong on a phase of respiration that improves the cranium's position; previously strong indicator muscles become weak when a phase of respiration makes the cranial position worse. When the phrase "a weak muscle associated with the cranial fault" is used, it refers to exactly that. All weak muscles are not necessarily associated with cranial faults.

Body Language of Cranial Faults

There are many factors which should alert the physician to the possibility of cranial faults. He should be constantly observing for one of the most common indications of cranial faults which takes place during routine evaluation of a patient when using manual muscle testing. The patient with a cranial fault will unknowingly take and hold a phase of respiration as the muscle test is done. This is the body's automatic effort to place itself in the best position for resisting the force of the muscle test. An individual with a cranial fault that is improved by thoracic inspiration will automatically take and hold an inspiration as resistance is developed to perform the muscle test. On the other hand, an individual with a cranial fault that will be improved by thoracic expiration will blow the air out and hold it while the test is performed. The physician should constantly be aware of this because such action nearly always indicates a cranial fault. The individual who does not have cranial faults is equally strong whether there is thoracic inspiration, expiration, or a neutral aspect of respiration.

More subtle body language of cranial faults is seen in the patient who does not take a phase of respiration and hold it; rather, he will hold respiration as it is when the test is performed. There are certain cranial faults where an individual is strongest in the central phase of respiration, either in or out.

When it is observed that a patient takes or holds a phase of respiration while muscle testing is being done, the physician should have the patient consciously breathe in a relaxed, natural manner. This is important because if cranial faults are responsible for the patient's health problem, his subconscious respiratory change will mask or hide the problem. To further test the effect of the patient's thoracic respiration on examination, the physician can have the individual take and hold the phase of respiration opposite that which is automatically taken; then a previously strong indicator muscle is tested for weakening. If it weakens, cranial faults are indicated and further evaluation should be done. A different form of test is to evaluate a weak muscle by having the patient take the phase of respiration which appears to enhance the muscle testing efforts and determine whether the weak muscle becomes strong — again, an indication of cranial faults.

The categories of body language of cranial faults relate to the following: (1) the type of health problem an individual has, (2) the history obtained during consultation, (3) asymmetry of the skull, and (4) remote structural health problems which influence the skull.

HEALTH PROBLEMS

Any health problem that potentially relates to cranial nerve control makes the cranial primary respiratory mechanism a candidate for evaluation. A thorough knowledge of structures supplied by the cranial nerves is important in making these observations.

Cranial faults are involved with many types of visual disturbance. The problem may be manifested as frank diplopia or difficulty in using the two eyes together as seen in "ocular lock," a type of neurologic disorganization recognized in applied kinesiology. Certain types of visual acuity problems respond very well to cranial corrections. Glaucoma is a condition that has been treated successfully by some in applied kinesiology.³ The procedure for all these conditions is simply evaluating and correcting the cranium in the usual manner. (A more thorough discussion of the eye and its relation to the cranium will be presented later.)

Conditions of the ear can manifest from disturbance of the auditory nerve, either the cochlear or the vestibular division. It is stated that cranial faults related to the temporal bone can disturb equilibrium by causing the right and left semicircular canals to be mechanically disoriented with each other.¹⁰ This appears to be accurate as evidenced by clinical results from cranial correction achieved in conditions of vertigo; it will sometimes relate with upper cervical correction, which apparently involves the tonic neck reflexes. Hearing loss is often immediately influenced by cranial correction, especially when the patient complains of a dramatic hearing loss after recent trauma, such as a "whiplash" accident.

Any type of neurologic disorganization may have associated cranial faults because of the great interplay between cranial nerves and the equilibrium mechanism of the body. The visual righting and labyrinthine reflexes may not be communicating properly with other organizational reflexes of the body. Structural imbalance resulting in postural strain, recurrent subluxations, and general muscular disorganization may result.

Conditions that relate directly with cranial nerves, such as Bell's palsy or trigeminal neuralgia, should receive cranial evaluation. There may be a functional problem which responds rapidly to cranial correction, or there may be a pathological condition which perhaps requires surgery. Differential diagnosis must be made in all conditions involving apparent cranial dysfunction.

Headaches always indicate the need for an examination of the cranial primary respiratory sys-

tem. There are many mechanisms by which cranial faults can create headaches, whether it be simple encroachment on the nerve within the suture or more complicated mechanisms. It is even possible for headaches to result from cranial faults on an indirect basis. There may be disturbance in a remote system, such as the digestive, which creates toxicity and in turn causes a toxic-type headache. The original problem is from cranial disturbance influencing the

vagus nerve; the end product is an indirectly caused headache.

There are many conditions which have been related to cranial faults, either through physiology or by observation. Some conditions have been empirically related to specific cranial faults. This association, although not constant, will be mentioned under "Conditions Correlating" in the discussion of each individual cranial fault.

HISTORY

A patient's history obtained during consultation may give clues of probable cranial faults. The prenatal and birth history — as well as observation of the newborn — can give valuable clues. A difficult birth, including long labor, type of presentation, improper use of forceps, and excessive sedation, may contribute to cranial faults. It is not unusual, when cranial faults are found in a child, to have the mother offer to bring in pictures taken of the infant at the hospital which reveal severe forceps marks and observable asymmetry of the skull.

An infant's activity and function are important to consider. An infant with cranial disturbance is often listless and has difficulty in nursing and swallowing. There may be visual disturbances, such as strabismus or nystagmus; digestive disturbances are common, with apparent allergic reactions to food.

As a child develops from infancy to early childhood, specific patterns are often present. During this early stage of development asymmetry of the skull is often easily observed, and there may be an improper closure of the six fontanelles. The child will often bump or rub his head against objects; this apparently is an effort to mobilize the skull. The most common evaluation by an applied kinesiologist is neurologic disorganization (switching), which is observed by poor development of mobilization on the floor. Later the child develops a poor crawling pattern (see Volume I).

When the patient is an adult, it is often difficult to initially obtain a history of birth, infancy, and early childhood, but the information frequently is forthcoming. The physician familiar with the cranial mechanism can usually detect that the dysfunction began early in life. If this is mentioned to the adult patient, a discussion with parents will often ensue. The patient will return with confirming information about his early history as related by a mother, father, or other relative.

Distortions that develop from birth or in early infancy and go uncorrected are more problematic

than faults which develop in the adult skull. The flexible infant skull, if distorted, sets the stage for development of asymmetry and possibly poor action of the primary respiratory mechanism.

It is important that an adult understand the significance of birth trauma to the cranium, because it frequently has an influence on the prognosis of a condition. If there is severe asymmetry, more time is usually required for correction. The amount of permanent correction that can be obtained may be limited.

Trauma after the skull has formed normally is more easily corrected and does not usually have the lasting effects which may be present as a result of birth trauma. The trauma may be directly to the skull — such as raising up underneath a cabinet, hitting the head on a car door, falling and striking the head, or blows of just about any nature. It is interesting that what sometimes appears to be an insignificant blow to the head can create more problems from cranial faults than what may appear to be severe trauma. It seems that the influence depends on whether the head is struck in precisely the right direction, with the exact phase of respiration that is conducive to the creation of a cranial fault. The skull can often accept severe trauma with no apparent disturbance. It is almost impossible to predict the amount of disturbance which will develop by evaluating the amount of force, velocity, direction, and area of impact.

Trauma creating cranial faults can be indirect. The so-called "whiplash" injury is a common cause of cranial faults. Many individuals who fail to recover from a seemingly innocuous injury do so because there is some problem in the cranium which has eluded detection. This includes the often seen digestive, visual, and auditory disturbances caused by the injury, and also neck pain, dizziness, etc. Another type of remote injury that frequently causes cranial problems — but is not generally considered as an injury to the cranium — is a sit-down type of

Examination and Treatment of Cranial Faults

fall, usually including direct injury to the sacrum. The sacrum is an intricate part of the primary respiratory mechanism and is discussed in Chapter 7.

A routine area of investigation for all physicians should be the stomatognathic system. Even though the oral cavity and the jaw have been considered the province of the dentist, it is necessary to include them in "total person" health care. Failure to do so will cause many significant factors to be overlooked. Questions should relate to symptoms of the stomatognathic system, such as jaw or tooth pain, bruxism (grinding of the teeth), thumb-sucking, and tongue-thrusting. A history of dental procedures will sometimes reveal a date coinciding with that of origination of symptoms.

Cranial faults can develop as a result of tooth extraction, the seating of a crown, improper occlusion, or dental appliances, as well as from other

procedures. The physician who has not previously investigated this area during history-taking will be surprised at the frequency of coinciding events. When the subject is brought up the patient will often say, "I asked my dentist if my new crown could be causing my eye problem, and he said no — it's just a coincidence." The dentist was being truthful in light of his current level of understanding.

It is necessary that all dentists become aware of the possible influence of dental procedures on the cranial primary respiratory mechanism, and evaluate the mechanism whenever symptomatic involvements coincide with a procedure. In fact, there should be routine evaluation of a patient for cranial disturbance before he is dismissed from dental treatment. Here again, an ounce of prevention is worth a pound of cure.

ASYMMETRY

Observing for lack of symmetry has two values in applied kinesiology. It provides clues through body language that cranial faults may be present, and it sometimes gives information regarding the type of cranial fault, thus reducing examination time. In applied kinesiology, evaluation of skull symmetry is not used as a final determination for the type of correction to be made.

Asymmetry is present either from imbalance of the bones on the two sides of the skull, or from an imbalance of the muscles. When asymmetry is a result of bone imbalance, it probably relates to birth trauma or injury in the infant or very young child when portions of the skull are still membranous or much more plastic than in the adult. These asymmetries may regain some balance with cranial correction. It is unlikely that complete symmetry will be obtained.

Lack of symmetry due to muscular imbalance may be secondary to asymmetry of the skull bones, or it may be primary. If this type of asymmetry is present, there will often be a return of balanced appearance to the skull. There is a thorough discussion of the muscles of the skull and the asymmetries caused by their imbalance in Chapter 4.

The accompanying pictures — showing first a natural picture and then one split to use half of the picture to make a full face — illustrate lack of symmetry in the skull and facial muscles. Note in 6—3 how the halves of the skull fail to match. The combined right halves cause the head to appear

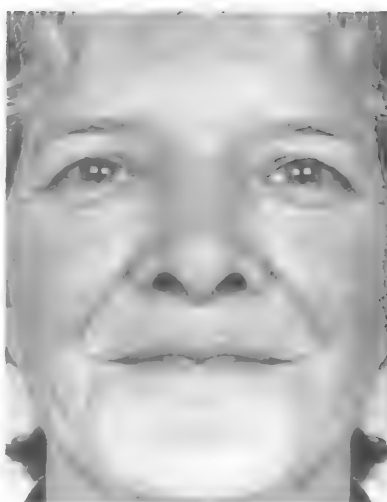
much larger, while the left sides appear narrower.

Functional imbalance of facial movement needs to be put into perspective. It may be due to an imbalance of the facial nerves controlling the muscles of expression, or it may be due to the characteristics of the individual's facial expression, developed as a part of the personality. Facial expression often relates to the balance of right and left brain activity. A comparison of facial muscle activity with other aspects of the cranial evaluation will put this into perspective. It is important to determine whether asymmetry is a result of bone or muscle imbalance, or a combination of both. As stated, bone asymmetry indicates chronicity from infancy or early childhood. Muscular asymmetry is important, not only in evaluating facial nerve function, but also because of a basic principle in applied kinesiology. It has long been observed that bone structural imbalance is caused by muscular imbalance. To make permanent correction of structural imbalance, it is necessary to restore normal function to the muscles. An imbalance of muscle function can give clues regarding the type of cranial fault present. This is discussed later with each individual cranial fault.

When observing for asymmetry, the patient is usually viewed from the straight A-P position. If photographs are taken for future reference, some method of aligning the skull so that the camera is in the center line must be available. Some texts show pictures demonstrating asymmetry which is actually photographic distortion. Close observation reveals



Total Face



Right Sides Combined



Left Sides Combined

6—2. Note the imbalance of the facial muscles as well as the mandible's deviation to the right. The nasolabial fold is deeper on the right. This may be due to dysfunction of the muscle or to bony asymmetry.

that the camera was slightly off-center and not level with the skull.

Often the clinical observation of asymmetry can be augmented by looking at the patient from a position where the face appears upside down. The examiner can stand at the head of the examination table, looking down at the supine patient, so that visualization of the face is upside-down. This helps make the observation one of structure, taking out of the picture the face's personality. We are accustomed to

looking at people from the normal point of view and therefore we do not observe asymmetries; rather, we equate them as part of the personality (which they may or may not be). Another way to objectively view the skull is to look at one portion at a time. Use a piece of cardboard or your hand to cover the mouth and nose while looking at the eyes and forehead. After using this method of observation, an ability to better see small deviations that are perhaps quite significant will eventually develop.



Total Face



Right Sides Combined



Left Sides Combined

6—3. The total face picture was taken with the external auditory meatus level and with no rotation relative to the camera. Note the eyes are not level in the total face; they are in the combined halves. The head is wider in the right side combination, and narrower in the left side combination.

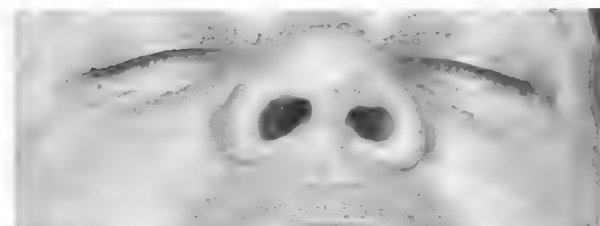
Examination and Treatment of Cranial Faults

Looking at the eyes for asymmetry is usually observing for protruded eyes in relation to a specific cranial fault. Two methods are used to easily identify eye protrusion. (1) Observe the almond-shaped opening made by the upper and lower eyelids rather than looking for an actual protrusion. The larger almond shape will be on the side of the more protruded eye. (2) Observe the eyes by looking at them from inferior to superior. This is accomplished by the examiner placing his head close to the abdominal surface while the patient is supine. A superior to inferior view can also be used. This is best obtained with the physician standing at the head of the table while the patient lies supine, and looking across the face toward the feet.



6—4. Left eye protrusion. Note the increase of visible upper eyelid; yet the almond-shaped opening is bilaterally close to the same size.

Relative nares size is significant in certain types of cranial faults. Look caudal to cephalad at the same time eye protrusion is evaluated. The open nostril seems to have a slight flare to it, as if the dilator naris muscle were contracting.



6—5. Nares imbalance.

The temporal bone has a motion of lateral flaring of the squama on sphenobasilar flexion. When a cranial fault includes excessive motion of the temporal bone in this position, the skull appears to have a bulge on that side. When the temporal bone is lesioned in the opposite direction, the skull appears to have a slight concavity. Typically, the skull will have a bulge on one side and a concavity on the other. This type of cranial fault — a temporal bulge — has been nicknamed “banana head” in applied kinesiology, referring to the banana shape of the head. Additional body language of the skull in this cranial fault is external flaring of the ear on the side of convexity and the ear of the opposite side closer to the skull.

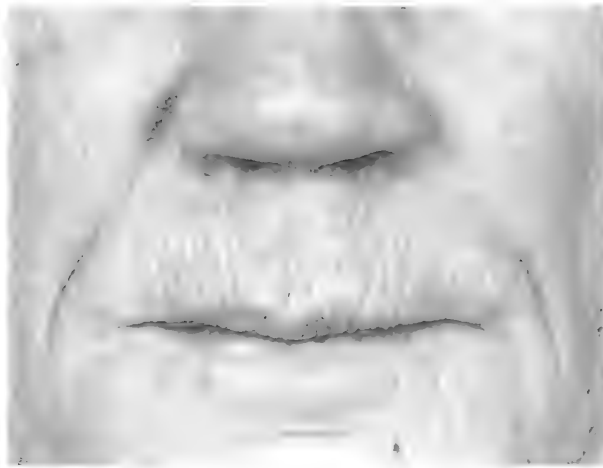


6—6. Imbalance indicating probable temporal bulge and parietal descent.

The frontal bone may show a greater amount of eminence on one side. This is usually related to an imbalance of the eyes, with one appearing more protruded. The horizontal lines of the forehead may be deeper on one side, with possibly even a lack of lines on the opposite side. This imbalance relates to the occipitofrontalis complex of muscles. Deep vertical lines at the glabella indicate activity of the corrugator supercilii.

The nose may appear off center line, which may be due to direct trauma. This imbalance can throw off the examiner's evaluation of the face, making it appear that the entire skull is asymmetrical when actually it is not.

The elevation and prominence of the zygomatic bone can provide clues of several types of cranial faults, as well as be related with temporomandibular joint dysfunction.



6—7. Deepened nasolabial fold. Lines indicate hyper-tonicity of orbicularis oris and muscles inserting into it.



6—8. Wider and higher zygomatic bone on the left. Probable eye protrusion on the left.

Balance of the orbicularis oris is influenced by the many muscles which have fibers intermingling into it. The nasolabial fold is made deeper by action of the levator anguli oris and zygomaticus major and minor muscles, of which the latter two cross the zygomatico-maxillary suture.

The position of the mandible and body language of the oral cavity will be discussed more thoroughly in Section II on the temporomandibular joint. For this evaluation, it is adequate to observe for the mandible's position in the center line. The line between the upper and lower central incisor teeth should be in alignment. When opening the mouth, the mandible should drop straight down without lateral deviation or popping of the temporomandibular joint.

Examination and Treatment of Cranial Faults

Posterior observation should reveal an occiput that appears balanced, with the head level on the neck and level with the shoulders. The occipital bone is formed from four ossification points. There is a significant possibility that the occipital bone can be malformed if there are cranial faults during its ossification. One condyle may be formed at a lower elevation than its opposite, creating a head-on-neck problem. An imbalance of the formation of a bone such as this is called an "interosseous cranial fault."

Another factor which should be evaluated in



6—9. Mandible to the right. Nares imbalance and stronger contraction of the zygomaticus major and minor muscles on the left.

reference to skull symmetry does not necessarily relate to the shape of the skull, but rather to the sutures themselves. Pain along a suture line indicates there is a probable cranial fault. This will sometimes be spontaneously reported by the patient; other times it must be found by the examiner's palpation. There is value in comparing the same sutures on both sides of the skull. While palpating the suture, the examiner should observe for an apparent separation or jamming. A separation is often palpable in older individuals who are thin and possibly have little hair; this is sometimes visually observable. In most cases it is necessary to palpate for the separation. A jammed suture feels like a ridge and may have a puffiness of the soft tissue. The most important factor is not whether the suture is jammed or separated, but rather the tenderness along it. The final determination of separation or jamming is noted by challenge, a further examination step which is explained later.

REMOTE PROBLEMS

There are specific types of remote health problems which consistently relate with cranial faults. Temporomandibular joint and pelvic function have already been mentioned, and both are discussed in detail later in this text.

Dysfunction of the gait mechanism, cloacal synchronization, pitch, roll, yaw, and tilt synchronization, and cervical spine dysfunction may all cause or perpetuate cranial faults. These are discussed in Volume I. Orthopedic problems, especially in the

feet, are also frequently associated with cranial problems and are discussed in Volume IV.

Whenever these remote problems are present, the cranium should be screened for possible involvement. There is often an interrelationship of these problems. The remote problem can create or perpetuate cranial faults as mentioned, or cranial faults can produce or perpetuate the remote problem. Again it becomes obvious that the body functions as a totally integrated whole.

Types of Examination

There are three primary approaches used in evaluating the cranial primary respiratory mechanism. Most systems concentrate on one approach. The first approach has been emphasized in the previous discussion on body language of cranial faults, i.e., observation for skull asymmetry. Another approach is astute palpation for motion of the primary respiratory mechanism. This was developed

by Sutherland¹⁵ and is used primarily in the osteopathic profession. The third approach uses manual muscle testing to evaluate function, and applies various factors to observe change in muscle strength for analysis of the primary respiratory mechanism. This is the approach developed by Goodheart⁵ and used primarily in applied kinesiology.

VISUAL OBSERVATION

The approaches in cranial therapeutics which use visual observation for symmetry work on the premise that a balanced skull is one which has good function. When asymmetry is observed, an effort is made to manipulate the skull to a more balanced position. This is a problematic approach because it does not take into consideration the possibility that the bones of the skull may be malformed due to stresses present during infancy and early childhood. Examination by various methods reveals that a skull which has considerable distortion may have no cranial faults causing disturbance in physiology. On the other hand, some skulls which appear to be symmetrical reveal considerable evidence of dysfunction, as observed by various methods of evaluation.

Attempting to force an imbalanced skull into symmetry may jam the sutures, distort normal function, and generally create iatrogenic problems. A system of evaluating actual function is the first criterion necessary for a viable approach in cranial therapeutics.

Although observing for lack of symmetry is not an optimum method within itself to determine any corrections needed for the cranial primary respiratory mechanism, it does provide valuable information. Visual observation indicates whether cranial faults are likely, and what type they could be. Further evaluation is necessary to confirm this and determine how a correction should be made.

PALPATION

Palpation is the major diagnostic factor used in osteopathy in the cranial field.¹¹ It is also used to a limited extent by other diagnostic approaches to the cranium. The osteopathic use of palpation is for position, determination of motion, and detection of lesions. A brief discussion of the procedures is to acquaint the AK practitioner with the method. The approach should not be attempted without thorough study and personal instruction.

Position

Palpation for position is used to augment visual observation. In cranial osteopathic palpation, gentleness — a very light touch — is stressed. Great care is taken in learning the art of this very discriminating type of palpation. In this approach to ultimately evaluate the position of the various bones of the skull and face, attention to soft tissue characteristics is emphasized. The muscles and fascia are evaluated for contraction, and the nature of the tissues is observed.

In evaluating bone position, it is necessary to have firmly in mind the motions made by the various bones in the primary respiratory mechanism. For example, on sphenobasilar flexion the tips of the mastoid processes move posteriorly and medially. A comparison of the right and left mastoid tips indicates the relative activity of the two bones. If the mastoid tip is posterior and medial on one side and anterior and lateral on the opposite, a three-dimensional cranial lesion is indicated. This will be discussed further under the specific types of cranial faults. This portion of the palpatory examination is regularly used in applied kinesiology, as well as in cranial osteopathy.

Motion

Motion evaluation in cranial osteopathy is for the cranial rhythmical impulse, involuntary active motion of the cranial bones, and passive motion. Again, all these approaches require very discriminating palpatory art. The admonition is continually given to use “gentle, tactile discrimination” with “feeling, thinking, seeing, knowing fingers.”¹¹

The cranial rhythmical impulse (CRI) is the general impulse of the cranial primary respiratory mechanism. It is the autonomous motion of 10-14 cycles per minute in the normal individual. It is independent of thoracic respiration, but influenced by it. There are specific palpation methods in cranial osteopathy for observing the timing of this impulse. It is felt over the asterion by use of the full palm of the hand.

Involuntary active motion: Involuntary active motion is motion of the individual skull bones with the cranial rhythmical impulse. This is the motion of individual skull bones during the two phases of cranial respiration — sphenobasilar flexion (inspiration) and sphenobasilar extension (expiration). In normal function, there should be specific movement of each bone with these cycles which corresponds to the cranial rhythmical impulse. It must be emphasized that the terms “inspiration” and “expiration” relating to sphenobasilar flexion and extension do not refer to thoracic respiration. These motions continue to occur when thoracic respiration is held. There is, however, a correlation with forced thoracic inspiration or expiration as observed in applied kinesiology and apparently in cranial osteopathy. Involuntary active motion of the bones of the skull is also called “physiologic motion.”

Passive motion: Passive motion is that which is induced by the operator. It is a system of diagnosis used in cranial osteopathy where the examiner directs light pressure to a skull bone to impart motion to the bone. The examiner then — with discrete palpatory skills — follows the motion of the bone, or lack of it. Magoun¹¹ emphasizes this point by describing the activity necessary to move a boat floating next to a dock. He points out that the boat goes nowhere if hit with a fist, but if gently pushed, it can be started into motion and then simply followed. This descriptively indicates the gentleness of action used in both examination and therapeutics in the osteopathic cranial field. The force used in applied kinesiology cranial correction is similar.

Palpation for Lesions

There are two methods listed by Magoun¹¹ to determine cranial lesions. These are alteration of permitted motion and alteration of cerebrospinal fluid fluctuation. Again, both methods require astute palpation and should not be attempted without specific training.

Alteration of permitted motion: With knowledge of skull motion, a single bone or group of bones is gently moved in a specific direction by the examiner's digital contact. Once the effort to move the bone(s) has been initiated, the examiner follows through with tactile perception to determine if movement is available and, if so, its range. The movement will either be accepted or rejected by the body. By this method normal skull motions are evaluated to determine if the skull's function allows the motion. The pattern of resistance in a skull may be logical and reasonable, considering all aspects, or

Examination and Treatment of Cranial Faults

there may be lack of logic in the individual bone's motion and position as a result of trauma; this is considered as "unphysiologic" motion.

Alteration of cerebrospinal fluid fluctuation:

This aspect of the examination evaluates for motion directed to a particular area of the skull by the examiner's digital pressure applied to another area. When a particular area is to be evaluated, light digital pressure is applied at the contralateral pole. For example, if the occipitomastoid suture is to be evaluated, the examiner evaluates both the occipital bone and the mastoid by placing one finger on each bone; then with the other hand he applies a light digital pressure to the frontal bone on the contralateral side. The hypothesis is that the pressure on the frontal bone directs the cerebrospinal fluid from the frontal bone diagonally across to increase motion at the occipitomastoid suture. A comparison of the occipitomastoid sutures is made by repeating the procedure on the opposite side. In the presence of a lesion, there will not be an increase in activity palpated at the occipitomastoid suture.

Palpation as a primary diagnostic tool for the evaluation of the cranial primary respiratory mechanism offers several advantages; it also has its disadvantages. The approach is an objective method if the examiner has developed skill in the procedure. Studies have shown both good and poor abilities from different individuals.^{13, 16, 17, 19} With this skill the approach to determine proper skull motion does not rely simplistically on the skull "not looking normal." It objectively evaluates which articulations do not have adequate motion, and indicates specific therapeutic efforts needed. The therapy used with this concept is gentle, and there are objective criteria for administration of the therapy; thus iatrogenic problems are minimal. After therapy has been administered, the skull can be re-evaluated objectively to determine if improvement has been obtained. The major disadvantage appears to be the considerable amount of art

required in obtaining accurate information from the palpation to make the above determinations. It should be obvious that various individuals will develop a wide range of proficiency in the use of this system.

Frymann has developed procedures presented in a monograph to learn the art of palpation.⁴ Her presentation, designed for a workshop, develops the art through stages of determining living structure from dead, feeling resilience, observing motion, and classifying different types of rhythms of body motion.

Upledger and Karni,¹⁷ in an effort to evaluate the palpatory ability of the osteopathic physician trained in the cranial field, measured body function by electromyography, electrocardiography, and strain gauge recordings of respiratory activity while an osteopathic physician trained in cranial palpation reported blindly his sensation of cranial activity. They found various activities of body functions which correlated with specific impressions the physician had of skull activity. They report the correlation far exceeds random probability; however, no statistical analysis was presented.

In another study by Upledger,¹⁶ interexaminer reliability on nineteen parameters of craniosacral palpation was evaluated. Overall agreement was 86%. The agreement on certain of the nineteen parameters was very high, while on others it was low. In a study by Roppel et al.,¹³ an apparatus was designed to give motion to a pair of artificial parietal bones. Both trained and untrained individuals "examined" for movement of the apparatus. Up to ten seconds of delay was allowed to take into account lack of response behind the movement. The results showed an inverse relation to accuracy and decision delay. The highest accuracy was reported by a subject trained in cranial palpation, but in some instances untrained individuals scored higher than those who were trained.

APPLIED KINESIOLOGY

The applied kinesiology approach to the cranial primary respiratory mechanism was developed by Goodheart⁵ and uses manual muscle testing as a system for evaluating cranial function. It evaluates the influences of the muscles of the cranium, face, and mastication on cranial primary respiratory function. If muscle imbalance is found, the standard applied kinesiology methods of muscle therapeutics are used. The AK approach also integrates total body activity, evaluating the interaction of the

cranial-sacral primary respiratory mechanism with other body functions. For example, cranial faults can be secondary to disturbance in any aspect of the triad of health. Structural disturbance may come from body imbalance from the foot upward. Certain chemical imbalances can cause cranial and pelvic faults, as can certain mental aspects.

To evaluate the cranial-sacral primary respiratory mechanism with applied kinesiology, it is necessary to have basic skills in AK. If the physician has not

studied muscle testing, therapy localization, challenge, etc., Volume I should be consulted.

Reproducibility of cranial evaluation regarding interexaminer reliability relates to the examiner's skill in manual muscle testing (see Volume I, Chapter 15).

Cranial examination in applied kinesiology uses change in muscle strength as perceived on manual muscle testing to evaluate function. As has been mentioned, the change in muscle strength may result from a specific phase of thoracic respiration. The thoracic respiration is interpreted to change the relation of the skull bones. Thus the change in muscle strength appears to relate to either improving function or being detrimental to it because of change in the cranial primary respiratory mechanism. Knowledge of the motion of the skull bones with thoracic respiration indicates the type of correction that may be necessary.

Therapy localization as used in phases of applied kinesiology is applied to the cranial primary respiratory mechanism. Therapy localization can be used to help determine when cranial faults are present, and if they have been corrected. Again, it is necessary to point out that positive therapy localization only tells something is wrong in an area. Positive therapy localization to an area of the skull does not necessarily indicate that a cranial fault is present, but combined with other diagnostic factors it is an important tool of investigation.

The challenge mechanism, combined with manual muscle testing, is the most valuable tool available in this form of cranial investigation. Challenge not only reveals what type of cranial fault is present; it gives specific information in most cases to determine the

Examination and Treatment of Cranial Faults

type of correction required, and whether or not the correction has been obtained after the therapeutic effort.

The applied kinesiology approach to the cranium specifically indicates how a correction should be made, thus reducing iatrogenic problems which may develop as a result of cranial therapeutics to a bare minimum. Most important is the ability to re-evaluate after therapeutic efforts to determine if correction has been obtained, or if the mechanism has possibly been placed into greater jeopardy.

Cranial therapeutics should not be attempted without training and knowledge of skull function. There are verbal reports of severe iatrogenic problems resulting from inept cranial manipulation. Clinical response and lack of iatrogenic problems have been extremely satisfactory following the applied kinesiology examination and therapeutic approaches described in this text. It is strongly recommended that the study of this text be augmented by personal instruction from a diplomate of the International College of Applied Kinesiology. The workshop procedures used in teaching the subject give the practical experience necessary to properly conduct the examination and administer the therapy. It is strongly advised not to use forceful, concussive manipulations on the skull. An investigation of the delicate, intricate structure of the various skull sutures indicates the need for accurate, gentle manipulative efforts. Concussive thrusts have the potential of damaging the sutural structure, creating adhesions which ultimately cause increased skull disturbance.

Format of Cranial Fault Examination and Correction

To get the most benefit from this section on examination and correction of cranial faults, it is necessary to have the anatomical and philosophical concepts of cranial physiology well in mind. The approach to the cranium is divided into three primary classifications of cranial faults. These divisions relate to the major motion of the structure involved in the fault. As knowledge of the cranial mechanism grows, it is important to remember that the cranial faults listed here are not individually unique; rather there is a continuum of cranial faults. Most dysfunctioning skulls probably have characteris-

tics of several faults; one or two are usually major.

The breathing pattern, therapy localization, and challenge are given for each cranial fault. These are the primary examination factors that differentiate cranial faults from each other, and help to differentiate them from pathologic conditions or improper function that is not applicable to this type of cranial therapeutics. Pain locations and correlating conditions will be discussed with each cranial fault, where applicable. Correction will be demonstrated with illustrations and pictures.

MECHANICS OF THE FAULT

The cranial mechanism appears to lose its motion in specific patterns. These patterns have been designated in applied kinesiology as various types of cranial faults. In describing the mechanics of the fault, an attempt will be made to relate the direction in which the bone(s) of the skull have excessively moved, where there may be a locking mechanism, and what muscles or body distortions may be involved in creating or perpetuating the fault. This description is designed to give an overview of the cranial fault.

The position of the bone(s) is hypothesized by examination, which uses the procedure described on page 87 to evaluate the motion of the skull bones in the primary respiratory mechanism. This information is correlated with clinical evidence of the vectors of force which return normal function to the skull. As improved methods of quantitating skull bone motion are developed, these hypothesized mechanics may need to be changed. Additional factors may be involved, such as harmonic vibratory activity influenc-

ing electromagnetic or other energy fields. Until further research is accomplished, the working hypotheses presented here appear to be effective on a clinical basis, producing results in health care which might otherwise be unattainable.

As discussed previously (page 139), the cranial primary respiratory mechanism is influenced by thoracic respiration, but it is also independent of it. The phases of thoracic respiration mentioned throughout this section will be referred to as the breathing pattern or as respiratory assistance. It will relate to inspiration, expiration, forced inspiration, forced expiration, breath held in the middle of inspiration, and breath held in the middle of expiration. There are considerations of the breathing pattern in addition to that of causing a weak muscle to strengthen or a strong muscle to weaken, as has been discussed. Various phases of breathing can be used to cancel positive therapy localization or challenge. In considering the various cranial faults, this will help determine the type of therapy to be administered.

MECHANICS OF CORRECTION

The mechanics of correction for some types of cranial faults are straightforward, requiring minimal description. On the other hand, some corrections are indirect, with the therapeutic force traversing numerous bones with various types of leverage and different activities at the sutures. For these more complex corrections, there will be a description of the closed kinematic chain.

The closed kinematic chain is influenced differently by various vectors of force. These variable forces bring into play the interrelationship between various types of cranial faults. An understanding of this

interrelationship is important for the physician in order to be able to evaluate the cranial mechanism and its proper relationship with his examination findings. Certain types of cranial faults usually appear in combination. If the examination does not reveal this correlation, further evaluation is necessary before proper correction can be obtained. The ideal approach is to correct a minimal number of cranial faults and obtain maximum correction. Often an examination will reveal numerous cranial faults; properly applied correction of one or a few will correct many.

THERAPY LOCALIZATION OF CRANIAL FAULTS

Therapy localization as described by Goodheart⁶ and used extensively in applied kinesiology is a very effective tool in evaluating for cranial dysfunction; however, one must understand therapy localization's capabilities and also its limitations. It indicates only that there is some form of dysfunction present at the point the patient is touching. Regarding the cranium, positive therapy localization could indicate a cranial fault; however, it could also indicate an active reflex point, such as a neurovascular reflex or

a cranial stress receptor, or a dysfunctioning muscle. The factor in the muscle could be a proprioceptor, trigger point, or fascia. Pathology, such as a sinus infection or tumors, could also be indicated by positive therapy localization.

These additional factors that therapy localize positively can usually be differentially diagnosed by using other techniques of applied kinesiology evaluation for cranial faults, such as challenge and respiratory assist. Positive therapy localization can

be further evaluated by respiratory assistance to eliminate the positive finding. Elimination by a breath held in indicates some type of cranial fault which correlates with excessive sphenobasilar extension. On the other hand, positive therapy localization eliminated by a breath held out indicates a cranial fault associated with excessive sphenobasilar flexion. Further evaluation can be obtained by correlating the findings of therapy localization and respiratory assist with challenge (explained later) of the various cranial structures.

The best therapy localization for cranial faults is observed on or as close to the involved sutures as possible. Most of the time there will be several points of positive therapy localization. This is especially true on a new patient who has not yet had cranial treatment, because many cranial faults are usually involved. This does not mean that all the cranial faults indicated by therapy localization need to be treated. When the key cranial faults are found and corrected, other (more secondary) faults will often automatically correct as a result of the closed kinematic chain of cranial bone function.

Therapy localization can help find cranial faults; it is also a valuable tool to use after therapeutic efforts to determine if the cranium was indeed corrected. There are times after cranial treatment that the positive therapy localization may be eliminated but other indications are not, such as pain along a suture line and positive challenge. In these cases, the cranial fault will usually return soon after the patient begins to ambulate and function routinely. The therapy localization was eliminated because there was enough

Examination and Treatment of Cranial Faults

correction of the condition to temporarily improve the dysfunction, but not enough for a permanent correction. Upon minimal stress, such as a cranial challenge, walking, chewing, or otherwise functioning, the condition returns. This may be because of failure to correct the cranial fault, or other cranial faults may still be present. The remaining problem may be muscular, in the form of imbalanced muscles specifically attached to or remote from the skull, or any of many possibilities. In other words, removal of positive therapy localization does not positively indicate correction of the cranial fault or contributing factors.

As each cranial fault is discussed, the areas of appropriate therapy localization will be mentioned. In some cases there will be numerous areas of therapy localization which will be put into perspective as to their level of efficiency for the specific cranial fault.

If positive therapy localization for a cranial fault, which is confirmed by challenge and other diagnostic factors, is not eliminated after therapeutic efforts but the challenge and other factors are, there is strong indication to further evaluate the patient for possible pathology or functional disturbance. The therapy localization could be positive as a result of the cranial fault, and also because of neoplasms, subdural hemorrhage, infection, etc. Continued positive therapy localization could also be a result of an active neurovascular reflex, stress receptor, or muscle proprioceptor. Failure to remove the positive therapy localization is strong indication that further evaluation is needed.

CHALLENGE

The best method for determining cranial dysfunction is the challenge mechanism (Volume I). This is used throughout applied kinesiology to evaluate articular function. Challenge refers to digital pressure applied in specific vectors, and observation for change in muscle strength observed by manual muscle testing. Challenge is the optimum approach in examination because it takes into consideration the individual aspects of the skull being examined. Moss,¹² studying the growth of the calvaria in the rat, inhibited growth areas to evaluate mechanisms of growth and influence on skull formation. During the growth period the suture position would shift, depending on the pressures applied to the skull. This caused individuality of sutures from skull to skull.

The use of challenge to find the correct vector of correction is somewhat more time-consuming than the previous method of simply pushing in certain

directions for specific cranial faults. The extra effort is actually timesaving because the very accurate correction of one cranial fault quite often corrects many faults through the closed kinematic chain.

There are two types of challenge. Rebound challenge, which is most frequently used, consists of placing a specific vector of force into an articulation, releasing the force, and then evaluating for muscle strength. Static challenge refers to placing a specific direction of force into an articulation and holding that force while observing for change in muscle strength. During these discussions, "challenge" will refer to a rebound challenge unless a static challenge is specifically described.

There has been considerable confusion about the challenge mechanism in applied kinesiology. This has come about primarily because previously written material did not delineate whether a static or a

Examination and Treatment of Cranial Faults

rebound challenge was being referred to; it was simply called challenge.^{14, 20} Since those writings, it appears that the best method is usually a rebound challenge. It has become the standard approach in applied kinesiology for the spinal column and skull, with few exceptions.

Cranial rebound challenge observes the body's reaction to a vector of force being placed into the articulations of the cranium. If the force does not relate to a cranial fault, the cranium and body will be oblivious to the challenge. If, however, the force does relate to a cranial fault, there will be a change in muscle strength on manual muscle testing. There are two types of muscular reaction. (1) A muscle which tests weak in the clear because of the cranial fault will become temporarily strong. (2) A muscle which tests strong in the clear and is not directly associated with the cranial fault will become temporarily weak.

A static cranial challenge requires the opposite vector of force to change muscle strength. This can be accomplished on some cranial faults and, at times, appears of interest for specific types of evaluation. An example of a static cranial challenge is pressing posteriorly on the mastoid process and holding that vector of force while a muscle is tested with no diaphragmatic respiratory change. If the cranial fault is an inspiration assist, this static challenge — held exactly correctly — will cause a muscle weak because of the fault to strengthen. The correction would be exactly opposite the direction of positive static challenge. The problem with using a static challenge is that there are variables present from the reciprocal tension membrane and movement of other bones. It is difficult to assess what would be an exact opposite direction to determine the method of correction.

Earlier in the development of applied kinesiology, the challenge mechanism of the cranium was not as thoroughly understood as it is today. In this discussion, there is considerably more detail about the types of challenge and their effects on the body. Most cranial correction in applied kinesiology's early days relied on indications of respiratory assist and therapy localization to locate the cranial fault. In this author's experience, the indications of therapy localization and respiratory assist can often be eliminated by *general* cranial manipulation; however, with accurate challenging, it will often be found that optimum cranial function has not been obtained. By using the challenge mechanism much more accurately in skull evaluation than was done in the past, more cranial faults will be found. The accurate use of challenge will also provide a higher degree of correction in the cranial mechanism.

The potential for iatrogenic problems resulting

from improper treatment is greatly reduced if treatment is provided only in the direction indicated by accurate challenge. Evaluating the challenge mechanism more thoroughly necessitates some change in the treatment approach. The vector of force used for correction must reproduce that indicated by the challenge.

The rebound challenge method of determining correction of cranial faults is ideal because it gives the exact vector of force needed. As the vector of force for the challenge is changed slightly, the previously strong indicator muscle weakens to varying degrees. There will be one specific vector which gives the maximum amount of change in muscle strength; slight variance from that vector will produce less change. The maximum change indicates when the exact vector of correction has been found. This enables corrections to be obtained without the hazard of reaction from improper treatment. Nearly all cranial faults are three-dimensional in nature; determining the direction of correction with only palpatory, visual, and breathing correlation does not give the information necessary to make the maximum correction and to avoid reaction.

The amount of pressure used for the challenge should be as similar for each challenge as possible. If the amount of force is changed when varying vectors are used, there is no way to compare challenges to find the best vector of force. The force used should be a light pressure, approximately one to four pounds. When the appropriate vector for correction is found, the light pressure will be adequate to cause a significant muscular strength change. In some very rigid skulls, a heavier challenge may be necessary.

On some occasions when the vector of force that caused a positive challenge is repeated, it will no longer be positive. This is because with minimal cranial faults, the challenge itself can be a corrective force. In this case, continue to evaluate the skull because the minimal cranial fault which was corrected by challenge is probably not the primary involvement.

The exact mechanism of the cranial challenge is unknown; probably many factors are responsible. This would account for the various types of challenges used in evaluating the different cranial faults for therapeutic measures. Among the areas that appear to be influenced by challenge are the reciprocal tension membrane and primary respiratory mechanism as described by Sutherland¹⁵ and DeJarnette,² the myelinated and unmyelinated nerves within the cranial sutures, and direct influence on the cranial nerves themselves. As additional information from research is obtained, more is being learned about this effective clinical tool.

MUSCLE CORRELATION

The muscles of the face and cranium — especially those of mastication — influence the cranium and may create, perpetuate, or correct cranial faults. The muscles influencing a cranial fault are listed, where applicable, but the list is not all-inclusive. It is sometimes necessary to treat these

muscles directly by attention to the muscle proprioceptors, or by use of the origin-insertion, fascial flush, and spray and stretch techniques. Where frequent clinical experience indicates a specific technique for a muscle, it will be mentioned.

PAIN LOCATION

Pain associated with cranial faults is often in the sutures. There may be generalized pain, such as that “behind the eyeball,” or pain in the muscles. Some cranial faults have become associated with specific pain patterns. This is from clinical observation and is by no means a pattern that will indicate whether a

particular cranial fault is actually present. Correction of the cranial fault will often dramatically reduce the pain, or eliminate it altogether. The pain patterns of cranial faults are primarily those of body language. They provide help to guide diagnosis, but they should not be relied on for final therapeutic determination.

CONDITIONS CORRELATING

Similar to pain patterns, various health problems have become associated with specific cranial faults. This, too, is a clinical observation and does not definitely indicate a cranial fault. The likelihood of a

cranial fault's presence is high; however, the specific fault may not be present. Another cranial fault might be responsible for the patient's health problem, or the cranium may not be involved at all.

CORRECTION

In all aspects of dysfunction, the body attempts to be a self-correcting, self-maintaining mechanism. Many cranial faults are never observed by the patient or doctor because the body corrects the problem before it causes symptoms. It is only when the dysfunction becomes locked and persistent that evaluation and treatment must be instituted. The objective in obtaining cranial correction is to impart a force into the cranium which will easily be accepted by the body for unlocking the mechanism and returning it to its dynamic state. In designing examination and corrective approaches, several goals should be met:

- 1) The corrective procedure should not assault the skull in a manner that would possibly create iatrogenic problems;

- 2) Evaluation of the problem should give specific information regarding how correction is to be obtained;

- 3) The corrective effort should achieve a very high percentage of corrections on the first attempt;

- 4) A system of re-evaluation immediately after the correction is attempted must be available to determine if the method used was successful;

- 5) The correction should be a lasting one, without need for further treatment of the same dysfunction;

- 6) The system's procedures should be quick to perform and easily reproduced by anyone trained in them.

1. Avoiding Iatrogenic Problems

Whenever efforts are made to change structure to affect function, it is possible to impart a force which does not improve function but, in effect, makes it worse. This, of course, is an iatrogenic problem, and all efforts should be made to avoid such accidents. The best method of making corrections is to ask the body, by using the challenge mechanism, what type of force it desires to obtain the correction, rather than to educationally decide what the body needs and then try to impose that decision on it.

It bears repeating that observing for asymmetry of the skull gives specific clues about the type of cranial correction necessary; however, this is similar to saying a straight spine is a healthy spine and then trying to force every spine into a straight position. The important factor is whether dysfunction is being

Examination and Treatment of Cranial Faults

caused by lack of symmetry. It is possible that the skull was out of balance during youth when it was extremely flexible, and it ossified in the imbalanced state but has normal primary respiratory function. Attempting to force this skull into symmetry could create great dysfunction of the cerebrospinal fluid mechanism, cranial nerves, circulatory function, etc.

Observing for discrete motion of the skull by palpation is an excellent way to find problems in the cranial primary respiratory mechanism for those highly trained in such astute observation; however, the location of impaired motion does not necessarily give information regarding what force should be imparted to the skull to unlock the mechanism. Without other tests to determine how to make corrections, the physician must be highly educated in the movement of each skull bone, angles of the sutures, and interaction between the bones. Even with this knowledge, corrective attempts will not be accurate for each skull because of anatomical variances. This is evidenced by the varying location of important structures such as pivot points, slightly different angles of sutures, remaining synchondroses, or progressive ossification and congenital anomalies.

Another factor which bears repeating is that no traumatic force should be used in an effort to correct cranial faults. The skull, regardless of its rigidity, will begin to function at its optimum level if gentle but persistent forces are applied in an accurate vector to help return it to normal function. "Persistent forces" does not mean many office calls; rather, in some cases it means considerable repetition, sometimes up to fifty or one hundred times before an active correction is obtained. Not always will correction be obtained in very rigid skulls on the first visit. Concussive forces designed to free the skull can cause damage to the sutures. These strong concussive forces may be designed by the doctor to "break the suture loose," but if that is the case trauma to the tissue only causes new adhesions, compounding the problem. First it is necessary to find an accurate vector of correction. A method which imparts a mild force to the skull and then observes the body's reaction to that force is desirable, because it actually asks the body what type of action is required for correction. This is what applied kinesiology challenge does. After determining the vector of correction, a gentle but firm pressure is applied with the correct phase of respiration and in the proper direction. The force has to be applied slowly and forcefully without concussion and with the correct phase of respiration. This amounts to working with the body, not against it. With this method, iatrogenic problems are minimal. In this author's experience with thousands of cranial corrections, there has been **no** known

iatrogenic problem, even temporarily. This cannot be said of other approaches which use forceful concussive efforts toward correction, or fail to make adequate analysis.

2. Specificity of Corrective Force

Since the body is a self-correcting, self-maintaining mechanism it will often accept a vector of force to aid it in unlocking the cranial primary respiratory mechanism, even though the force is not an ideal effort. This is fortunate, because sometimes the body will take accidental forces and make corrections out of them. In some instances, improved cranial function can be obtained by applying a force exactly opposite that described for correction in this text. Most of the corrections described here are rebound-type; they have been clinically found to be the most effective for consistent corrections. A therapeutic effort should provide the ideal force with the exact phase of respiration for optimum correction. In applied kinesiology evaluation, the cranium reacts to many vectors of challenge; however, there will be one specific vector which creates maximum change in muscle strength on manual muscle testing. This is the specific correction which should be used.

3. High Percentage of First Effort Correction

Correction with first effort is evidence the evaluation system is effective in finding and categorizing the problem. Obtaining this correction depends upon several factors. The specificity of effort as outlined above is required, along with the use of the exact phase of respiration which is optimum for the body to help make the correction. Another factor is the doctor's expertise in evaluating cranial motion. With experience, the doctor will be able to feel the cranium begin to move with the treatment force when correction is being obtained. Sometimes only a few repetitions are needed and movement begins. On other occasions, when there is great chronicity or other factors causing considerable locking of the skull sutures, many repetitions of the corrective force may be needed. If the doctor is knowledgeable about the exact vector of force required and can feel skull motion, he can continue his therapeutic effort without fear of iatrogenic problems until the skull yields to the force. The doctor experienced in cranial therapy rarely has to apply a second effort to correct the cranial fault; however, the fault may return at a later time if there are other factors involved with the problem which have not yet been corrected (see #5).

4. Re-evaluation Ability

The system of cranial evaluation should have

many methods of evaluating whether the therapeutic attempt was successful and, further, evaluating on future visits whether the correction is holding. Naturally, the more factors of re-evaluation, the better feedback the doctor has regarding his corrective procedure. Evidence of a correction in applied kinesiology is elimination of positive therapy localization, challenge, and reduction of palpatory findings and pain locations. Muscles weak because of the cranial fault should test strong on manual muscle testing after correction, and there should be no weakening on the various phases of breathing.

5. Lasting Correction

The same evaluation procedures that originally found the fault are used during subsequent visits to evaluate whether the correction has held. Since the cranial primary respiratory mechanism is only one part of a totally integrated body, it may possibly not hold its correction because another portion of the body is transmitting stress to it. This could be pelvic imbalance, foot dysfunction, cervical muscle imbalance, or a dysfunction of the masticatory system, to name only a few. If a cranial fault returns, the doctor must look further to find which of the innumerable factors is causing the return.

Examination and Treatment of Cranial Faults

6. Easily Reproducible Corrections

The system of cranial examination and correction should not require great amounts of skill (an acquired art). It should be easily reproducible by anyone trained in the procedures. In applied kinesiology, the major requirement is that the doctor be knowledgeable in the science and art of manual muscle testing. Of course, this is a requirement in all phases of applied kinesiology. The application of applied kinesiology to the cranial primary respiratory mechanism is relatively easy to learn in comparison with other systems of cranial therapeutics. Naturally, as more expertise is developed, the corrections are more efficiently accomplished and contributing dysfunction is more readily found. The important factor is that if the physician follows the indications given by observing the body's desire for correction, iatrogenic problems are greatly minimized. The primary requirement for the doctor new to cranial evaluation and correction is to remember to use gentle pressures, not forcing the cranium, but rather working with it and using the vectors of force and respiration indicated by examination. Do not attempt to move the cranium on only the apparent indications of lack of symmetry, pain locations, and symptomatic complaints.

Flexion-Extension Group of Cranial Faults

The flexion-extension group of cranial faults refers primarily to those which have improper or lack of movement about the transverse axis in the coronal plane. This relates primarily, then, to sphenobasilar flexion and extension movement. Cranial faults in this

group are the inspiration and expiration assist, the sphenobasilar inspiration and expiration assist, and the glabella cranial fault. In this discussion, it is important to recognize the difference between the mastoid tip, process, and portion.

INSPIRATION ASSIST CRANIAL FAULT

The inspiration assist cranial fault is characterized by a muscle weak in the clear becoming strong when the patient holds an inspiration. The fault is also characterized by a previously strong indicator muscle weakening when the patient holds a full phase of expiration.

Inspiration and expiration cranial faults often relate to a large number of other cranial problems. Proper correction of these faults may eliminate many other cranial faults. This is usually the most advantageous mechanism to approach first when beginning cranial correction.

Examination and Treatment of Cranial Faults

Mechanics of the Fault

The inspiration assist cranial fault occurs when the skull is too far into sphenobasilar extension; consequently, when the subject takes a deep breath in and holds it, a muscle which is weak as a result of the cranial fault becomes strong. This is because the inspiration brings the sphenobasilar junction toward flexion (out of lesion).

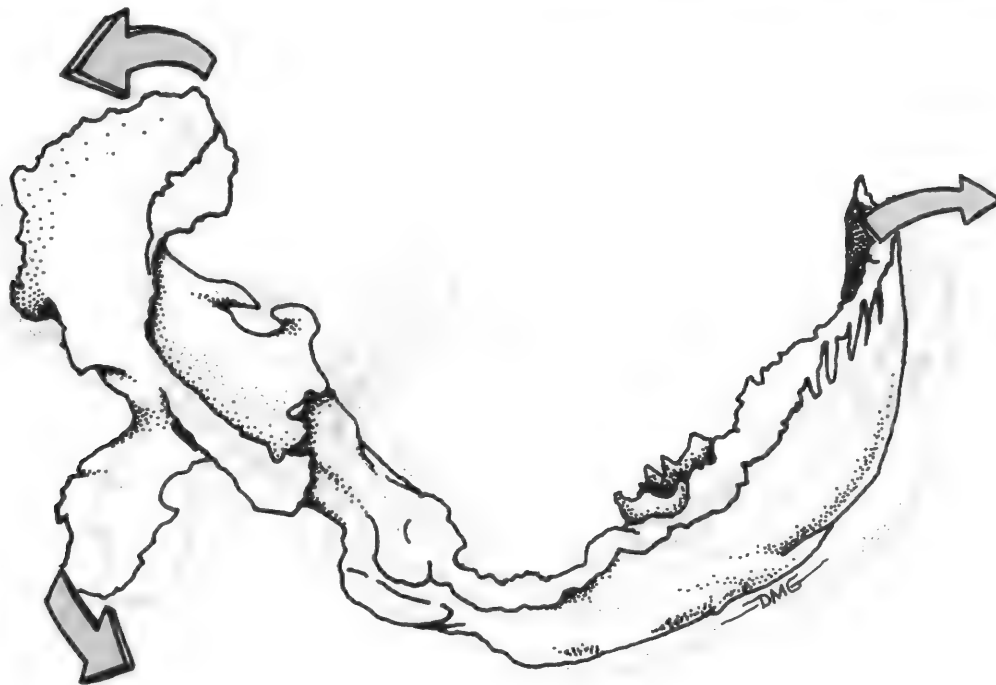
The abnormal position of the occipital bone is with the upper squamous portion anterior and superior, and the basilar portion inferior. There may or may not be rotation of the occipital bone about the sagittal axis, or lateral bending at the sphenobasilar junction; thus there may be a three-dimensional distortion, making challenge necessary to find the exact direction of correction.

the mastoid process are anterior and slightly lateral. The upper squamous area is usually internal and posterior, while the lower squamous area below the zygomatic process is anterior and medial. The exact position of the squama and mastoid process depends on the amount of rotation and side-bending of the sphenobasilar junction about the sagittal axis.

This cranial fault may be unilateral or bilateral. Each side should be examined independently. If bilateral, the two sides may be similar or different in the exact position of the fault.

Mechanics of Correction

The mechanics of correction use an inspiration assist by the patient and a mechanical vector to increase the cranial fault, thus unlocking it and



6—10. The solid black lines represent an exaggerated sphenobasilar extension which is present in an inspiration assist cranial fault. The gray lines and arrows indicate movement of the sphenoid and occipital bones toward normal position with inspiration. Arrows indicate general motion only.

The sphenoid is inferior at the posterior aspect of the body. The anterior portion of the floor of the greater wing is anterior and slightly superior, while the superior portion of the lateral wall of the greater wing of the sphenoid is posterior. The pterygoid processes are anterior and slightly medial and superior.

The temporal bone is very much involved in this cranial fault as a part of the closed kinematic chain. With no other cranial faults to complicate the temporal bone's position, the mastoid portion is posterior, while the mastoid process and the tip of

allowing the reciprocal tension membrane and muscles to correct the position. The most advantageous vector of force is determined by challenging to see which vector causes the maximum amount of muscular change. Observing the muscular change is best done by using a previously strong indicator muscle and noting its weakening during challenge. The force is applied, in general, to the posterior aspect of the mastoid process. The more inferior the contact on the mastoid process, the better leverage obtained on the axis of rotation of the temporal bone.

The vector most often found to give the maximum change with challenge is that of pushing on the posterior, slightly medial aspect of the mastoid process. This vector tends to increase internal flare and posterior position of the upper portion of the squama of the temporal bone, which appears to be the usual position of the temporal in an inspiration assist cranial fault. The optimum vector can vary considerably because of the three-dimensional aspect of most cranial faults. The challenge will take into account whether there is sphenobasilar torsion in the sagittal plane, or side-bending in the transverse plane; these are described later. Sometimes it is also necessary to introduce an inferior or superior vector of force into the mastoid process for optimum challenge.

The mechanics of motion imparted to the occipital bone and sphenoid by temporal bone movement are very interesting. As anterior pressure is applied to the posterior aspect of the mastoid process, there is a separation of the occipitomastoid suture below the condylosquamomastoid pivot. Above the pivot, where the temporal and occipital bones interdigitate as the teeth of two gears, an opposite rotation of the two bones takes place. The occipital bone at this section moves downward and somewhat posteriorly, while the mastoid portion of the temporal bone moves downward and somewhat anteriorly (see figure 6—11).

A vector of force on the posterior aspect of the mastoid process imparts motion to the sphenoid via the temporal bone's articulation with the sphenoid below the sphenosquamous pivot. The inferior portion of the temporosphenoidal suture along the floor of the greater wing of the sphenoid is well below the general axis of rotation of the sphenoid bone. Thus, when the vector of force is applied to the posterior aspect of the mastoid process, the temporal bone pushes the floor of the greater wing of the sphenoid anteriorly, with force being applied through the inferior temporosphenoidal suture. The pterygoid processes move anteriorly with this motion. The superior aspect of the lateral surface of the greater wing moves posteriorly, since it is above the sphenosquamous pivot. The widely beveled squamous suture of the superior lateral surface allows a gliding motion between the sphenoid and temporal above the pivot. Structures posterior to the pivot, such as the clivus and sphenobasilar junction, move inferiorly (see figure 6—12).

Pressure on the posterior aspect of the mastoid process increases sphenobasilar extension. When medial or lateral vectors of force are introduced on the mastoid process, an influence on the internal or external position of the superior squama of the

Examination and Treatment of Cranial Faults

temporal bone develops. The temporal bone, as a connection between the occipital and sphenoid bones, is very important in many complexes of cranial faults. When there is torsion or side-bending of the sphenobasilar junction, as well as extension or flexion, the three-dimensional correction on the mastoid process can correct all three factors of the distortion.

Therapy Localization

When an inspiration assist cranial fault is present, there will be positive therapy localization in many areas. One of the best locations to observe is along the occipitomastoid suture. Usually there will also be positive therapy localization along the temporo-parietal suture (squamosal suture) and temporo-sphenoidal suture.

Positive therapy localization by itself is not adequate information for diagnosing an inspiration assist cranial fault. Many other cranial faults, as well as other factors, can cause positive therapy localization in these areas.

Challenge

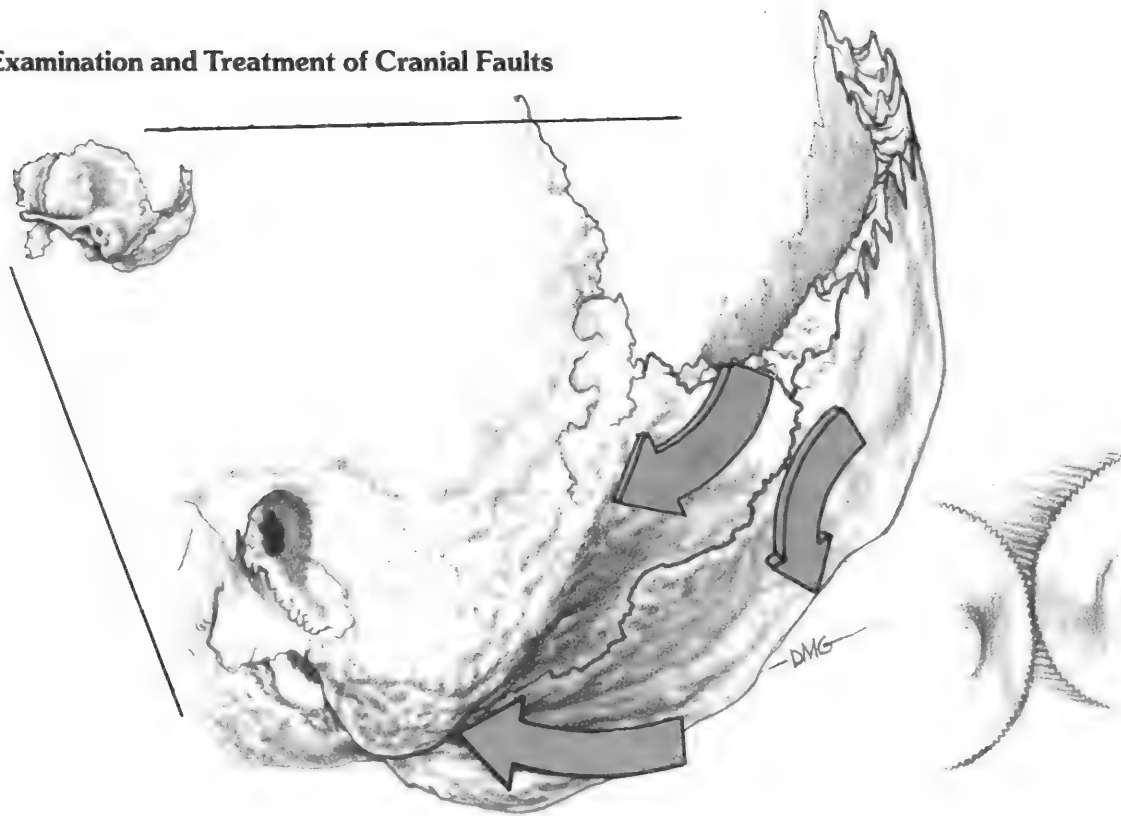
When an inspiration assist is suspected, the doctor applies a mild force (one to two pounds) on the posterior aspect of the mastoid process. After releasing the challenge pressure, a previously strong indicator muscle is evaluated for weakening. The vector is changed until the maximum weakening of the indicator muscle is perceived. This is the best direction for correction.

Challenge in an anterior, slightly lateral direction is that which is most often found. Often the challenge will vary greatly from the usual. To obtain maximum weakening of the indicator muscle, the challenge may require a significant medial, lateral, superior or inferior component. In this case, there is probably some other cranial fault(s) which tends to be the primary involvement. Correction of the inspiration assist cranial fault with an accurate vector will often correct the other cranial fault(s). In any event, effective correction of the inspiration assist fault will make the other fault(s) easier to correct.

Muscle Correlation

Numerous muscles may be either hypo- or hypertonic to create or perpetuate the inspiration assist cranial fault. This large amount of muscular involvement is found because the inspiration — as well as the expiration — assist cranial fault in one way or another relates with nearly all other cranial involvements, and with temporomandibular joint dysfunction.

Examination and Treatment of Cranial Faults



6—11. Challenge or therapeutic pressure on posterior aspect of the mastoid process moves the bones in the direction of the arrows. The area above the condylosquamomastoid pivot moves as interdigitation of teeth on two gears.

The primary postural muscles directly involved are the sternocleidomastoid and the upper division of the trapezius. Their imbalance may be directly contributory to the cranial fault, or may be secondary to cranial dysfunction. These muscles receive their primary afferent nerve supply from the spinal accessory nerve, and efferent supply from the ventral rami of the 2nd, 3rd, and sometimes 4th cervical spinal nerves. Imbalance of these muscles should alert the physician to consider remote structural involvement, such as foot or pelvic dysfunction, organization of the body as evaluated by PRY technique (Volume I), and cervical subluxations, as well as other — more infrequent — factors.

Intrinsic muscles of the skull which may be involved are the temporoparietalis, auricularis anterior, superior and posterior, occipitofrontalis, and the muscles of mastication.

The muscles of mastication are the temporalis, masseter, and internal and external pterygoids, with some contribution from the buccinator. These muscles have great influence on skull function and are discussed thoroughly in Section II of this text.

Pain Location

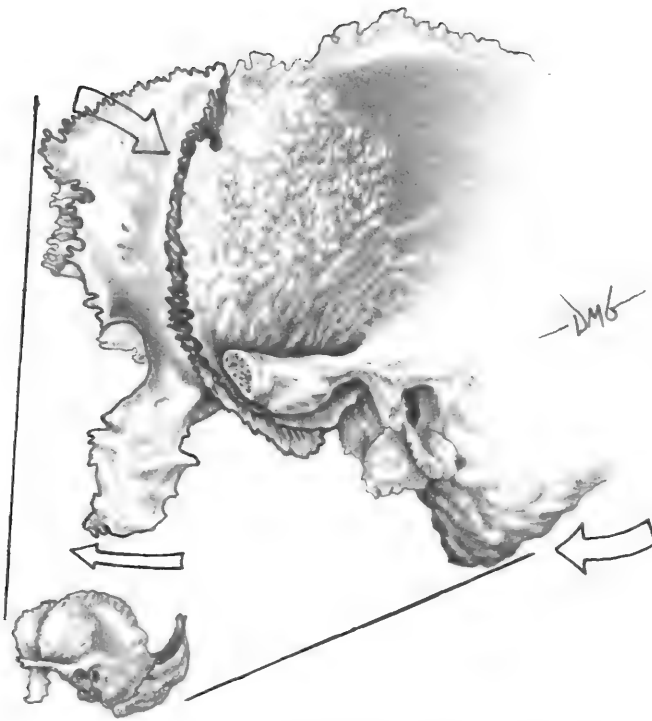
Although pain is not a consistent indicator for this cranial fault, it is often good to evaluate for its presence to help determine improvement after

correction. Pain will often be located on the frontal bone along the mid-pupillary line to the hairline. This is usually elicited by the examiner's digital pressure. Also, there will often be pain along the occipitomastoid suture, and sometimes along the coronal suture. The mastoid process and tip are often very tender to digital pressure.

Correction

Correction is obtained by applying approximately the same amount of force used for challenge in exactly the vector that produced the maximum change upon challenge. The force is applied while the patient takes a complete inspiration. It is important that the inspiration be from complete expiration to full inspiration, with the doctor applying the force throughout the phase, specifically at the end of respiration. The patient's respiration should be slow and deliberate, about 12 cycles per minute. Occasionally, a patient will inhale rapidly and must be slowed down by instructions from the physician. Usually four or five inspirations are adequate to obtain correction. If the mastoid process area is very tender, decrease the amount of pressure being applied and increase the number of repetitions.

Stabilizing the head on the opposite side with a broad contact helps direct the treating pressure exactly where the doctor wants it. Failure to stabilize



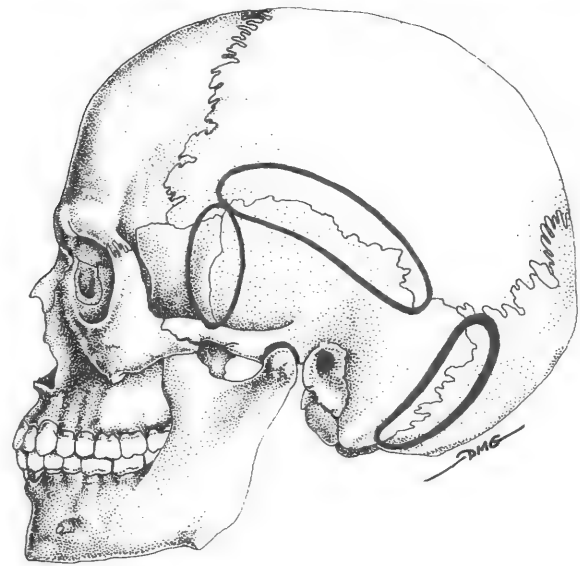
6—12. Challenge or therapeutic pressure on posterior mastoid tip directs force through the lower aspect of the temporosphenoidal suture to rotate the inferior aspect of the sphenoid anteriorly, increasing sphenobasilar extension. Sliding action takes place above the SS pivot.

allows the patient's head to roll slightly with the corrective pressure, somewhat minimizing its effect. The stabilization should be broad and accomplished in such a manner that it does not interfere with motion which may develop in sutures remote from the corrective force.

The patient will often feel the force transmitting through the skull. The sensation may be felt anywhere but is most often observed in the bony orbital, nasal, and maxillary areas. The more accurate the vector of corrective force, the more often a remote sensation will be felt by the patient. The sensation is generally not painful, but it may be slightly uncomfortable. Usually it is a sensation of movement or activity, difficult to describe.

The experienced doctor will feel the cranium begin to yield as the corrective force is applied. This movement indicates that correction has been obtained. Cranial movement can be felt by the doctor with almost all cranial corrections. It is a subtle movement that the physician should attempt to recognize. It is the best indicator of how many repetitions of therapeutic force should be applied to the skull.

After the corrective effort, re-evaluate with challenge in different vectors and with therapy localization. Test for improvement of muscles which were weak prior to the corrective attempt and were



6—13. Best therapy localization is obtained at the occipitomastoid suture; other areas indicated may show positive therapy localization.

strengthened with an inspiration held in, indicating that their weakness was associated with the cranial fault.

Tenderness associated with the cranial fault, as listed above, is usually significantly reduced or eliminated following successful correction. This reduction is a good indicator of the effectiveness of the correction.



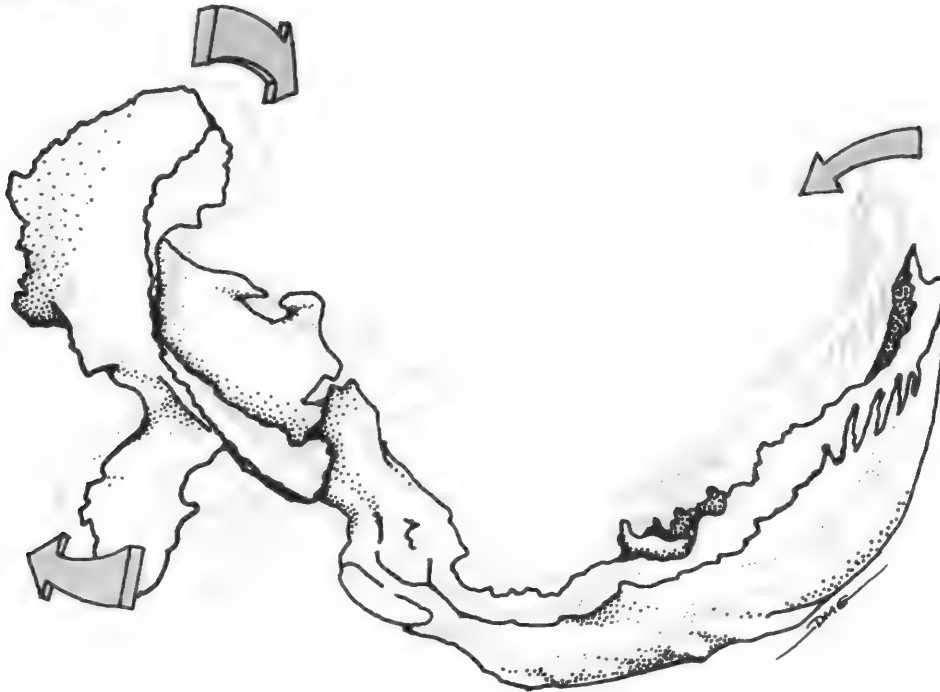
6—14. Pressure is applied for inspiration assist in the direction indicated by maximum weakening of an indicator muscle during challenge.

EXPIRATION ASSIST CRANIAL FAULT

The expiration assist cranial fault is characterized by a muscle which is weak in the clear because of the cranial fault becoming strong when the patient holds a full exhalation. A previously strong indicator muscle becomes weak when a full inspiration is held. This cranial fault does not occur as often as the inspiration assist. It is generally unilateral, with the opposite side of the skull being in an inspiration assist cranial fault. A bilateral expiration assist is occasionally found.

Mechanics of the Fault

The expiration assist cranial fault occurs when the skull is too far into sphenobasilar flexion. Since expiration brings the sphenobasilar junction toward extension, there is an improved relationship of the skull with exhalation; thus a muscle weak as a result of the fault will become strong when this phase of respiration is held.



6—15. The solid black lines represent an exaggerated sphenobasilar flexion which is present in an expiration assist cranial fault. The gray lines and arrows indicate movement of the sphenoid and occipital bones with expiration, bringing the bones toward normal position. Arrows indicate general motion only.

The expiration assist cranial fault is basically opposite the inspiration assist cranial fault. The malposition of the bones, challenge, and correction all apply to this opposite activity. The abnormal position of the occipital bone in this fault is a slightly posterior and inferior position of the superior portion

of the squama, with the basilar portion superior. As with the inspiration assist cranial fault, the occipital bone may be rotated along its sagittal axis or twisted into a lateral bending in reference to the sphenoid, giving the three-dimensional aspect to the fault.

The sphenoid primarily rotates about its coronal axis so that the posterior aspect of the body is superior. The anterior floor portion of the greater wing is posterior and slightly inferior, while the superior portion of the lateral wall of the greater wing of the sphenoid is anterior. The pterygoid processes are posterior.

As with the inspiration assist cranial fault, the temporal bone is very much involved. The squamous portion of the temporal bone will typically be lateral, the tip of the mastoid process will be posterior and medial, and the mastoid portion will be anterior.

Mechanics of Correction

Force is applied into the cranial mechanism through the temporal bone. The force is on the anterior aspect of the tip of the mastoid process, in a generally posterior direction, upon the patient's exhalation. The force is designed to increase the

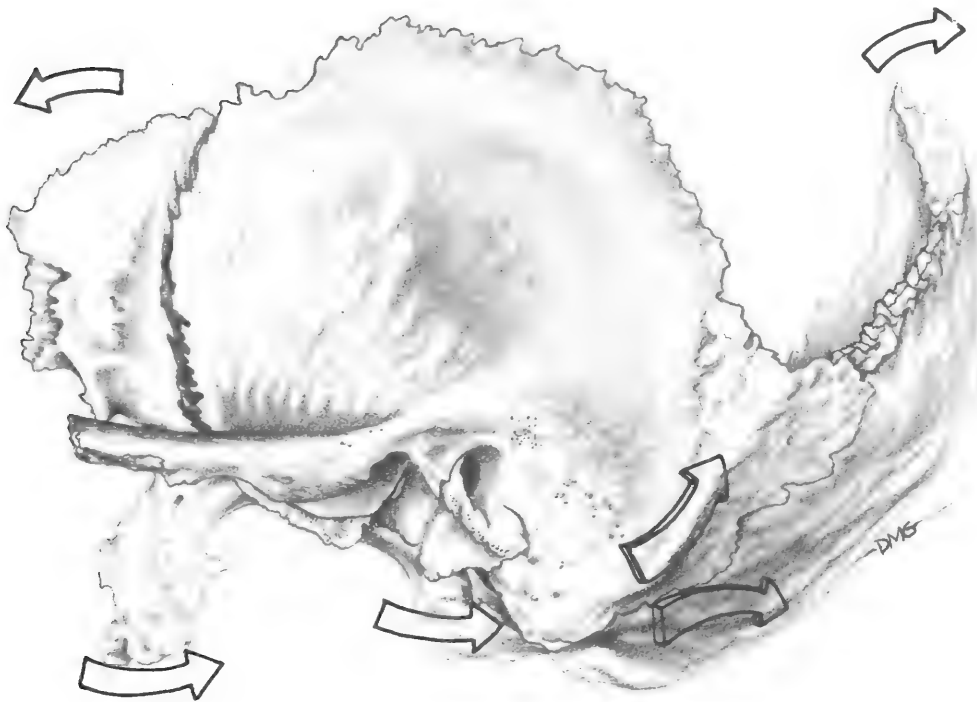
Examination and Treatment of Cranial Faults

Therapy Localization

The location for therapy localization is the same as for the inspiration assist cranial fault; it is usually best found along the occipitomastoid, temporo-parietal, and temporosphenoidal sutures. Since inspiration and expiration assist cranial faults are often combined with others, therapy localization in this area is not a positive diagnosis for the type of cranial fault. Also, it must be remembered that there are stress receptors, TS line points, and other factors in these areas which can show positive therapy localization.

cranial fault's mechanical position, thus unlocking the mechanism so that the primary respiratory mechanism — which includes the reciprocal tension membrane — may return normal function. The excessive sphenobasilar flexion is increased primarily through the gear-train type of suture between the temporal and occipital bones above the condylo-squamomastoid pivot, where there is an opposite rotation of the two bones.

As the temporal bone rotates, the temporo-sphenoidal suture below the zygomatic process



6—16. Pressure on the mastoid tip in the direction of optimum challenge causes rotation of the temporal bone to impart force on the sphenoid and occipital bones to increase sphenobasilar flexion.

moves posteriorly; this may pull the inferior portion of the sphenoid posteriorly, thus also increasing sphenobasilar flexion because the structure below the zygomatic process is inferior to the general axis of rotation of the sphenoid and moves posteriorly with sphenobasilar flexion. The posterior articulating surface of the floor of the greater wing of the sphenoid articulating with the temporal bone makes up this portion of the temporosphenoidal suture, the movement of which is primarily expansion and contraction.

Challenge

The expiration assist cranial fault is challenged by pressure applied primarily to the anterior aspect of the tip of the mastoid process in a posterior direction. After releasing the challenge pressure, the physician tests a previously strong indicator muscle for weakening. The vector of challenge is varied until the direction causing the greatest weakening of the indicator muscle is obtained. The challenge may have a medial, lateral, superior or inferior component in addition to the basic posterior direction of challenge.

Examination and Treatment of Cranial Faults

When a lateral vector is added to the challenge, it is sometimes necessary to "dig" into the tissues to contact a more medial aspect of the mastoid process. This requires short fingernails, and may be very uncomfortable to the patient. Determining the best direction of challenge is very important in obtaining maximum effectiveness of the therapeutic force to be applied during correction.

Muscle Correlation

As with the inspiration assist cranial fault, there are many muscles which can be involved with the expiration assist cranial fault. The primary postural muscles are the sternocleidomastoid and upper trapezius. The therapeutic indications are the same as those described earlier in reference to the inspiration assist cranial fault.

The longus capitis, with its insertion into the inferior surface of the basilar portion of the occipital bone, may be weak, allowing excessive sphenobasilar flexion. This muscle appears to be treated by the flexion and extension treatment of the atlas and occiput as described in Volume I.

Intrinsic muscles of the skull which may be involved are the temporoparietalis, auricularis anterior, superior and posterior, and the muscles of mastication. The muscles of mastication and general temporomandibular joint function must be considered in any cranial fault which involves the temporal bone. This, of course, means that almost any time the cranial mechanism dysfunctions, the temporomandibular joint and the muscles of mastication must be evaluated.

Pain Location

Pain locations and the indication for their evaluation are the same as for the inspiration assist cranial fault.

Correction

Corrective force is applied to the anterior aspect of the tip of the mastoid process in a generally posterior direction. The vector of force is the same one which caused a previously strong indicator muscle to weaken to its maximum. It is important that the exact vector observed in challenge be used. The maximum positive challenge will often have a lateral to medial component. If the challenge has a



6—17. Expiration assist therapeutic pressure may be applied straight posterior, as illustrated, or with a medial or lateral vector, depending on challenge.

medial to lateral component, the contact for correction is often difficult to obtain. As with challenge, it may be necessary to dig somewhat into the tissue to obtain good contact on the medial aspect of the tip of the mastoid process.

In both the inspiration and expiration assist cranial fault corrections, the patient often feels a sensation which radiates to the maxilla or frontal area, or other more generalized locations. The sensation may be described as a light pain, or simply as an unusual feeling. This is generally a good indication that the vector of force is influencing the skull on a broad basis. It will be observed that a slight vector change away from that indicated by maximum challenge will cause this sensation to disappear.

The therapeutic effort should be repeated with the patient's exhalation from total inspiration to total expiration, until the physician feels motion in the skull from the pressure being applied. When a physician is first beginning to work with the cranial mechanism and has not yet learned to feel skull motion, he should do four or five repetitions of the pressure with expiration, then evaluate to determine if the correction has been obtained. In any case, after it is expected that correction has been obtained, the patient should be re-evaluated with challenge, therapy localization, and pain locations to confirm the correction. By following the procedures outlined, especially those of accurate challenge and examination after correction, iatrogenic problems are negligible in cranial therapeutics.

SPHENOBASILAR INSPIRATION ASSIST CRANIAL FAULT

The sphenobasilar inspiration assist cranial fault is characterized by a weak muscle strengthening with a forced inspiration held in. "Forced inspiration" means the patient takes in as much breath as he can and then attempts to increase the inspiration even more. Forced expiration causes a previously strong indicator muscle to weaken. An inspiration assist sphenobasilar cranial fault is equivalent to what has been called a "sphenobasilar extension fault," inasmuch as the sphenobasilar junction is too far into extension. It is the same type of cranial fault as an inspiration assist; however, it is more securely locked in position and requires greater respiratory assistance and mechanical effort to unlock.

Mechanics of the Fault

The mechanics of the sphenobasilar inspiration assist cranial fault are identical in the cranium to those of the inspiration assist cranial fault. It appears that the only difference between the two is a more solid locking of the structures in lesion, and possibly the additional involvement of the facial bones.

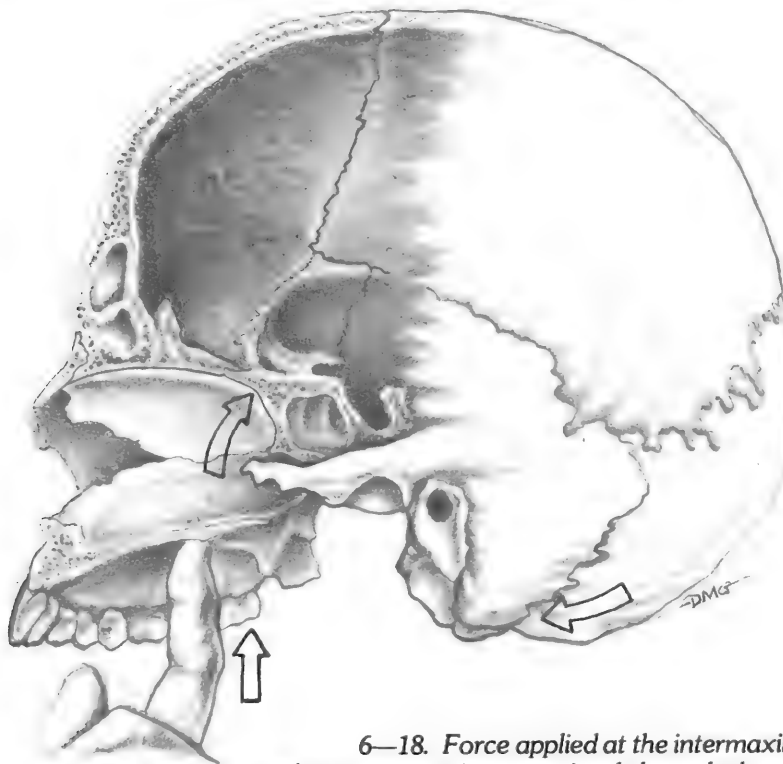
The position of the occipital, sphenoid, and temporal bones is exactly the same as in the inspiration assist cranial fault; thus the mechanics of the fault and its correction remain the same

regarding the interrelationship of these three bones. The additional factor is the relationship of the three bones mentioned with certain facial bones.

When the sphenoid and occipital bones are in excessive extension, the rostrum of the sphenoid moves superiorly and somewhat anteriorly. The vomer and the intermaxillary and interpalatine sutures also move superiorly and slightly anteriorly.

Mechanics of Correction

The corrective effort to the occipital and temporal bones by contact on the tip of the mastoid process is the same as that used in correcting an inspiration assist cranial fault. The additional factor in the mechanics of correction is an intra-oral contact on the palate. The contact is usually in the general location of the cruciate suture, with pressure applied in a generally superior direction. Force is transferred from the intermaxillary and interpalatine sutures through the vomer to the rostrum of the sphenoid, thus causing its superior motion. Superior motion of the rostrum of the sphenoid causes an inferior movement of the sphenobasilar junction, thus increasing its extension. Again, the correction is obtained by increasing the lesion to unlock the mechanism, thus allowing normal function.



6—18. Force applied at the intermaxillary or interpalatine sutures is transmitted through the vomer to the rostrum of the sphenoid, increasing sphenobasilar extension. Simultaneous pressure is applied on the posterior mastoid tip in an anterior direction determined by challenge.

Examination and Treatment of Cranial Faults

Therapy Localization

Therapy localization for the sphenobasilar cranial faults is the same as that for the inspiration and expiration assists; usually positive therapy localization at the cruciate suture is present, especially if there is thoracic respiration at the time of therapy localization. Challenge and the forced breathing pattern of this cranial fault are necessary to confirm the diagnosis.

Challenge

Challenge of this cranial fault requires a two-handed contact. One hand applies a force to the tip of the mastoid process similar to that used to correct the inspiration assist cranial fault.

The second contact is intra-oral in the general area of the palatomaxillary suture. Generally the pressure is directly superior. This should be determined by challenge, using various vectors of force until the maximum weakening of a previously strong indicator muscle is observed. The wide range of possible vectors observed on the tip of the mastoid process is not usually seen at the palatomaxillary suture. It will generally be a straight superior vector, with only slight deviation that will give the maximum challenge.

Usually challenge to the tip of the mastoid process and to the palatomaxillary suture can be done independently. It is sometimes of value to do a simultaneous challenge to the two areas. When this is done, it is best to first challenge the mastoid

process and find the optimum vector there; then continue to use that vector while applying various vectors to the palatomaxillary suture area, ultimately finding what two vectors give the maximum weakening of a previously strong indicator muscle. This is the optimum combination of forces to correct the fault.

Muscle Correlation

In addition to the muscles listed for the inspiration and expiration assist cranial faults, the muscles of the mouth are often involved. A sphenobasilar inspiration assist (sphenobasilar extension fault) which is subclinical is often brought into the clear by having the patient contract muscles which apply pressure to the buccal and labial surfaces of the upper dental arch. This tends to narrow the arch, forcing the intermaxillary and interpalatine sutures further superior and increasing the cranial fault by force transmitted through the vomer to the rostrum of the sphenoid. The facial muscle contraction is maintained with forced expiration, while a previously strong indicator muscle is tested for weakening to find hidden sphenobasilar inspiration assist faults.

Correction of muscular imbalance is very often accomplished by correcting the cranial faults and/or temporomandibular joint balancing as discussed in Section II. It may be necessary to use proprioceptive treatment to the muscles to generally induce relaxation. In some instances, the muscles may be weak and require strengthening.



6—19. Maintain contraction of muscles of the mouth and the buccinator while patient holds forced expiration to find a hidden sphenobasilar inspiration assist fault.

Pain Location

Pain location is the same as that described for an inspiration assist cranial fault, with the possible addition of pain on digital pressure at the cruciate suture. There may be a headache "behind the eye" as described by the patient. These additional pain locations vary from patient to patient.

Conditions Correlating

The sphenobasilar inspiration assist cranial fault may be bilateral or have a sphenobasilar expiration assist on the opposite side. When there is a sphenobasilar inspiration assist on one side and a sphenobasilar expiration assist on the other, they are treated independently.

Correction

Pressure is applied to the tip of the mastoid process and to the palatamaxillary suture area simultaneously as the patient goes from full expiration to full inspiration. The pressure is applied with four or five inspirations, or until the physician feels movement in the skull from his therapeutic pressure. In some very rigid skulls it may take ten, fifteen, or twenty repetitions. If the physician has not developed an ability to detect skull movement, it is best to re-evaluate after every four or five repetitions to determine if correction has been obtained.



6—20. Pressure is applied simultaneously on the two areas in the direction of maximum challenge, which was determined together or independently for each area.

Confirmation of correction is obtained by a combination of therapy localization, challenge, and observation of the strengthening of the muscles which were weak because of the cranial fault. A previously strong indicator muscle may also be tested while the patient holds a forced expiration. The forced expiration test of an indicator muscle can be enhanced by having the patient contract the muscles of the mouth, including the buccinator; this applies pressure to the buccal and labial surfaces of the teeth as previously described.

SPHENOBASILAR EXPIRATION ASSIST CRANIAL FAULT

The sphenobasilar expiration assist is similar to the expiration assist cranial fault; however, it is locked more securely, as is the sphenobasilar inspiration assist. The sphenobasilar expiration assist cranial fault is locked in sphenobasilar flexion; therefore it is identical to what has been called a "sphenobasilar flexion fault."

The sphenobasilar expiration assist is characterized by having the patient exhale to the maximum and then attempting more exhalation. This is termed "forced exhalation." Forced inhalation will cause a previously strong indicator muscle to weaken.

Mechanics of the Fault

With the sphenobasilar junction in excessive flexion, the sphenoid, occipital, and temporal bones are in a position similar to an expiration assist cranial fault. Like the sphenobasilar inspiration assist cranial fault, this fault is characterized by more significant involvement; it may also involve facial bone function.

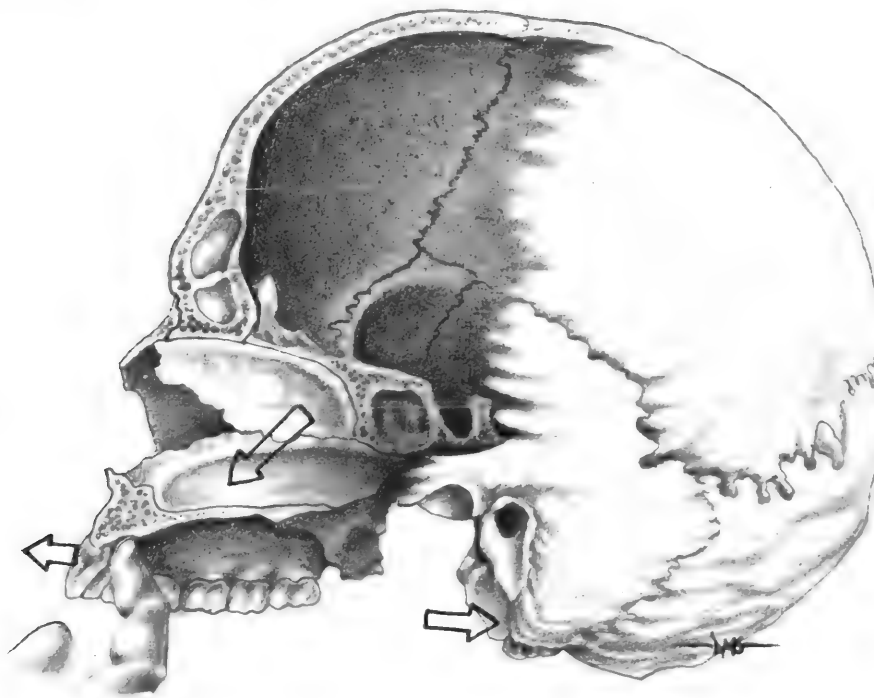
In excessive sphenobasilar flexion, the rostrum of the sphenoid is rotated inferiorly and somewhat

posteriorly. The vomer, connecting the sphenoid to the maxillae and palatine bones, is also inferior. This causes the intermaxillary and interpalatine sutures to be inferior, with the maxillary dental arch flared externally. The pterygoid processes are posterior and slightly lateral and inferior.

Mechanics of Correction

Correction of this cranial fault requires a two-handed contact. One hand is placed on the anterior aspect of the tip of the mastoid process, while the other contacts the central incisor, pulling it basically anteriorly. The contact on the mastoid process influences the cranium in the same manner as in the correction of an expiration assist cranial fault.

The anterior force applied to the central incisor usually includes some lateral direction which is determined by challenge. This force accentuates the external flare of the dental arch, lowers the intermaxillary and interpalatine sutures, and moves the rostrum of the sphenoid inferiorly by way of the vomer. This action increases sphenobasilar flexion,



6—21. Direction of pressure on the mastoid process is determined by challenge the same as for an expiration assist correction. The anterior portion of the sphenoid is pulled anteriorly and inferiorly by anterior pressure on incisors to increase sphenobasilar flexion for a rebound into extension. Connection from the maxillae to the sphenoid is by way of the vomer.

thus increasing the fault for a rebound correction after the mechanism is unlocked.

It is possible to have a bilateral sphenobasilar expiration assist, or a sphenobasilar expiration assist on one side and a sphenobasilar inspiration assist on the other. If the involvement is bilateral, both sides can be corrected at the same time; however, it is usually better to correct one side at a time because more accurate vectoring can be obtained. If an individual has a sphenobasilar inspiration assist on one side and a sphenobasilar expiration assist on the other, they should be corrected individually. The side having the more positive challenge is the one which should be corrected first; it is usually the sphenobasilar inspiration assist cranial fault. Its correction will sometimes correct the other side through the action of the closed kinematic chain of the cranium.

Therapy Localization

Therapy localization is the same as for an expiration assist cranial fault, usually with the addition of positive therapy localization at the cruciate suture. Therapy localization at the cruciate suture will more often be positive if the patient goes through full phases of respiration while therapy localizing.

Challenge

Challenge can be accomplished with either a two-handed challenge simultaneously, or by challenging each area separately. When separately challenging, the tip of the mastoid process is challenged from anterior to posterior in the same manner as an expiration assist cranial fault. The challenge at the central incisor is done by the physician contacting the central incisor and pulling it generally in an anterior direction, then testing a previously strong indicator muscle for weakening. The vector is changed with a medial, lateral, and sometimes superior and inferior direction in conjunction with the anterior direction. One particular vector will cause maximum weakening of the indicator muscle; this is the best vector for applying corrective force.

Sometimes it is necessary to simultaneously challenge at the tip of the mastoid process and the central incisor. This is a more complicated challenge because of the combination of vectors that can be applied when both are being tested at the same time. It is usually easier to first determine the optimum challenge at the tip of the mastoid process and to use this vector while challenging the central incisor with various vectors. In this way, the optimum double challenge can be obtained.

Muscle Correlation

Sometimes evidence of a sphenobasilar expiration assist cranial fault is enhanced by having the patient contract various facial muscles while holding a forced inspiration, while the examiner tests a previously strong indicator muscle. The muscle correlation does not seem to be as prevalent as in the sphenobasilar inspiration assist cranial fault.

Pain Location

Pain is often located at the occipitomastoid suture, and sometimes along the coronal suture. There may also be pain at the cruciate suture on digital pressure. Evaluating pain on digital pressure prior to correction is of value for comparison after corrections have been obtained. Generally there will be a notable reduction in the pain level upon the same digital pressure.

Correction

Correction is obtained by simultaneously pressing on the tip of the mastoid process and pulling on the central incisor in the direction that caused the maximum weakening of a previously strong indicator muscle on challenge. Therapeutic pressures are applied as the patient goes from complete inhalation to complete exhalation. The force should be applied lightly, working with the skull and not attempting to force it. The therapeutic pressure is repeated with exhalation four or five times, or until the physician feels motion in the skull from the pressure. Removal of positive therapy localization, challenge, and pain, and strengthening of the muscles which were weak as a result of the cranial fault, are indications that correction has been obtained.



6—22. Vectors are applied in the exact direction of challenge which caused the maximum weakening of a strong indicator muscle. Challenge may be accomplished one area at a time, or simultaneously.

COMBINED INSPIRATION AND EXPIRATION ASSIST CRANIAL FAULTS

A relatively common finding when the skull is evaluated with careful accuracy is inspiration assist and expiration assist cranial faults on opposite sides of the skull. This pattern is very important in obtaining optimum correction of the cranium; it may be overlooked if a cursory examination is done.

As previously stated, most cranial faults are three-dimensional in character, having components of flexion or extension in the sagittal plane and rotation about the coronal and sagittal axes. The cranium is often distorted so that one side of the skull accommodates distortion on the other. In other words, in an inspiration assist on one side and an expiration assist on the other, the squamous portion of the temporal bone will flare medially on the former side and be lateral on the latter side.

The combined inspiration and expiration assist cranial faults are often associated with neurologic disorganization, discussed in Volume I. Correcting these faults with accurate vectors determined by challenge will often remove the indicators for neurologic disorganization — such as positive therapy localization at K27 and “ocular lock” — with no specific therapeutic efforts applied to them.

Cranial mechanism influence on neurologic disorganization can be observed by having the patient with neurologic disorganization therapy localize to K27, causing a previously strong indicator muscle to weaken. An assistant then applies a static challenge to the cranium. When the correct vectors of force are obtained — usually indicating an inspiration assist on one side and an expiration assist on the other — the muscle which weakened as a result of the K27 therapy localization will regain normal strength. Also, an individual who exhibits ocular lock in the clear will not show that condition when the same static challenge is held on the cranium. Even though a static challenge indicates improved function, it is best to use a rebound challenge to determine the direction of correction. The static challenge used as a method for evaluation of correction does not allow the freedom of the reciprocal tension membrane and dynamics of the closed kinematic chain to actively indicate the correction needed.

After correction of the cranial involvement the patient should be evaluated for pelvic dysfunction, discussed thoroughly in Chapter 7. There will often be a category I pelvic fault. After its correction, the elimination of the positive therapy localization at K27 will be enhanced. This is probably due to the adaptive torque pattern of the shoulder girdle to

pelvic distortion.

The common association of cranial and pelvic distortion with neurologic disorganization is reasonable because of the considerable number of equilibrium proprioceptors involved. Apparently cloacal synchronization is often disturbed by pelvic dysfunction, and the cranial mechanism disturbs the organization of the visual righting and labyrinthine reflexes with other equilibrium proprioceptors. Probably the equilibrium proprioceptors located throughout the spine are also involved as a result of the distortion between the cranium and pelvis. It is not unusual to observe that upper cervical subluxations spontaneously correct after cranial fault correction. This is probably due to the relief of stress of the dura mater and of muscular balancing as a result of improved function of cranial nerve XI.

Mechanics of the Fault

The mechanics of this combination cranial fault are the same as those discussed under inspiration and expiration assist cranial faults. The skull appears to be in a twisted position, resulting in sphenobasilar flexion on one side and extension on the opposite; thus the squama of the temporal bone will be flared on the flexion side and medial on the extension side.

All aspects of a cranial examination usually correlate. If there is an inspiration assist fault on one side and an expiration assist on the other, they frequently correlate with other cranial faults that may be found. For example, a temporal bulge cranial fault, which is explained later, should be found — if present — on the same side as the inspiration assist cranial fault because the temporal bulge relates with inspiration. A parietal descent cranial fault, also described later, should be found — if present — on the expiration assist side. The significant amount of skull twist when this condition is present complicates the visual appearance, making it impossible to determine the types of faults present by looking for asymmetry. This may be explained as more is learned about the internal mechanisms of the skull, including the reciprocal tension membrane.

Mechanics of Correction

The mechanics of correction of these simultaneous cranial faults are the same as when they are present individually.

Therapy Localization

Therapy localization is found bilaterally and is the same as if the faults were found individually.

Challenge

It is important to find the exact vector of correction. This is easily accomplished when each mastoid tip is challenged individually. There will usually be a medial vector on one mastoid process and a lateral vector on the other in addition to the anterior or posterior vectors; however, this is not a consistent finding. Actual challenge must be done to find the optimum direction of correction.

Correction

As the patient takes a deep inspiration, pressure is applied to the tip of the mastoid process on the inspiration side, in the appropriate direction indicated

Examination and Treatment of Cranial Faults

by challenge. When full inspiration is reached, the patient exhales completely as the physician applies pressure to the expiration assist side in its appropriate direction. Generally, it is best to use a finger or thumb on the mastoid tip, while the palms of the hands cradle and stabilize the skull. Care must be taken that the stabilization does not restrict skull motion and retard the therapeutic effort.

After apparent correction has been obtained, re-evaluate for confirmation. Indications of neurologic disorganization from cranial disturbance — if present — should be abolished after correction, as should be positive therapy localization and challenge; any associated weak muscles should be strong.

GLABELLA CRANIAL FAULT

An individual should be able to breathe through his nose or mouth without creating problems. The glabella cranial fault is characterized by a patient's inability to breathe, either nasally or orally, without causing apparent dysfunction of the cranial primary respiratory mechanism. When the glabella cranial fault is present, a previously strong indicator muscle will weaken when the individual takes a deep inspiration — either orally or nasally — and holds it; it will not weaken with both types of inspiration.

Mechanics of the Fault

Involvement appears to be primarily that of the relationship of the cranial bones with flexion and extension. Because of this cranial fault's unusual relationship with breathing, the bones making up the nasal cavity appear to be significantly involved. In all probability, the frontal, ethmoid, vomer, palatine and maxillary — as well as the sphenoid and occipital bones — are the primary bones of involvement.

Most often the indicator muscle will weaken on an oral inspiration. When it weakens on nasal inspiration, there appears to be a correlation with trauma to the nose. In this case, the correction is the same as for oral weakening; it is simply done on oral inspiration rather than on nasal.

A second step in correcting this condition requires activation of the atlas, axis, and 3rd cervical, which indicates the probability of an involvement of the dura mater. The dura mater attaches to the rim of the foramen magnum, the axis, and the 3rd cervical; then it is primarily free until it attaches to the sacrum.

Mechanics of Correction

As with other cranial faults, the glabella cranial fault is often corrected as a result of correcting other faults, with no treatment given specifically for it. It does seem to require a higher percentage of individual correction than some of the other faults.

Contact for correction of this fault is two-handed, with one hand at the glabella and the other at the external occipital protuberance. The two hands apply force toward each other coincident with the type of inspiration — either oral or nasal — which did *not* cause the indicator muscle to weaken. Since the pressure applied to the occipital bone at the external occipital protuberance is only slightly higher than the general axis of bone rotation, it appears that its activity is primarily that of stabilization. The pressure applied to the glabella area generally appears to influence the frontal, ethmoid, sphenoid, maxillary, vomer, and palatine bones.

The second step of correction appears to influence the synchronization of movement of the upper cervical vertebrae with the skull.

Therapy Localization

Positive therapy localization is best obtained with the patient's two-handed contact. One hand is on the glabella and the other over the external occipital protuberance. Sometimes there will be positive therapy localization over the glabella only, but it is not as correlative to the cranial fault as two-handed therapy localization. As usual, therapy localization does not positively indicate that the fault is present. Accurate diagnosis is made with a combination of therapy localization, the breathing pattern, and

Examination and Treatment of Cranial Faults

challenge. Therapy localization is of value before and after a therapeutic effort to determine if correction has been obtained.

Challenge

Contact points for challenge are the same as those for therapy localization. The physician contacts the glabella with one hand and the external occipital protuberance with the other. Pressure of three to four pounds is applied at the two points toward each other. When the pressure is released, a previously strong indicator muscle is tested for weakening.

The glabella cranial fault does not appear to require challenge to determine the exact vector of correction. The therapeutic approach is generally done after other cranial faults — inspiration and expiration assist, sphenobasilar faults, etc. — have been corrected. These appear to take out the cranial rotational factors prior to correction of the glabella fault; in fact, correction of the other cranial faults first often eliminates the glabella cranial fault without specific therapeutic effort directed toward it.

Muscle Correlation

The nose muscles are often involved, particularly

the procerus and the corrugator supercilii. These are typically hypertonic and need to be relaxed with proprioceptive treatment. Balancing these muscles often improves a patient's ability to breathe, which has been restricted because of interference to the nasal passages. Body language of hypertonicity of the procerus and corrugator supercilii is either horizontal or vertical folds producing wrinkles at the glabella area.⁹

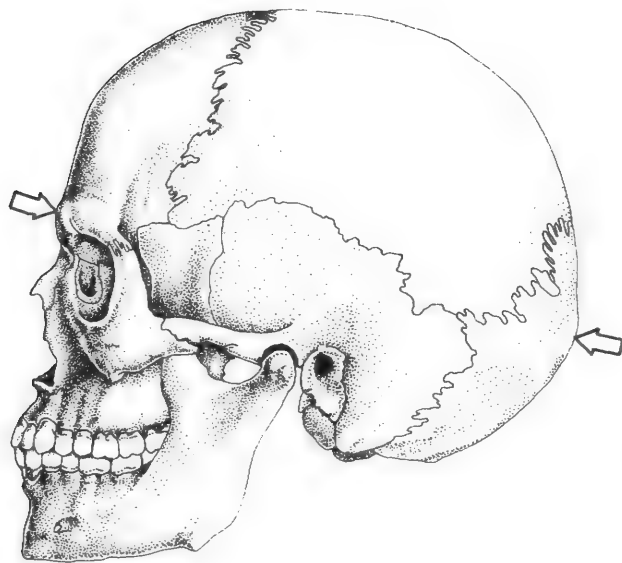
Pain Location

There does not appear to be any specific pattern of pain with this cranial fault.

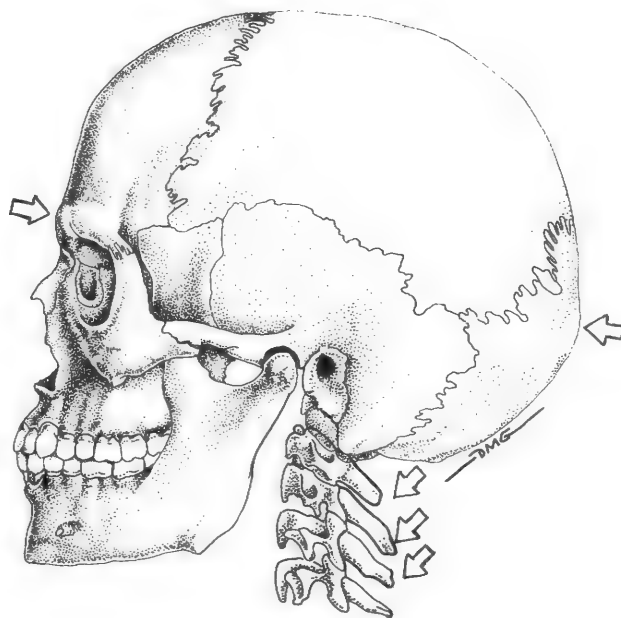
Conditions Correlating

The glabella cranial fault is often associated with blood pressure deviations, with hypertension being more common. Correction of the fault will often reduce diastolic blood pressure in idiopathic hypertension 10-20 mm Hg immediately, shown by re-testing after the fault has been corrected.

Approximately 60% of the time there will be a sacral fault with the same respiratory pattern. Evaluation and correction of the sacrum is discussed thoroughly in the following chapter; however, there is



6—23. Direction of force applied in first step of correcting the glabella cranial fault. The major activity appears to be at the glabella area; the force at the occipital bone is more for stabilization.



6—24. Second step in correction of the glabella cranial fault is to simultaneously move the posterior aspects of the upper 3 cervicals inferiorly.

a specific correlation to sacral correction with this cranial fault. With the patient prone, test the hamstrings on both nasal and oral inspiration. If the sacral fault is present, it should be on the same oral or nasal inspiration as the cranial fault. Correct with anterior pressure on the sacral apex on the oral or nasal inspiration which did not weaken the hamstrings. Repeat four or five times, then re-test.

Correction

The physician stands superior to the supine patient. With one hand he contacts the external occipital protuberance and with the other, the glabella. Pressure of the two hands is applied toward each other on either the oral or nasal inspiration that did not cause a strong indicator muscle to weaken. The pressure is applied four or five times; the number is usually indicated by the skull feeling as if it is beginning to yield to the pressure.

After completion of the first step, the physician changes the contact on the external occipital protuberance to the 5th digit, and uses the index, middle, and ring fingers to contact the 3rd, 2nd, and

Examination and Treatment of Cranial Faults

1st cervical vertebrae respectively. Contact is on the posterior arch of the atlas and on the spinous processes of the 2nd and 3rd cervicals. Pressure on the external occipital protuberance and glabella is continued as before with the same type of inspiration. Simultaneously the digits contacting the cervical vertebrae move them inferiorly. This is repeated four or five times.

After correction of the glabella cranial fault, re-evaluate with challenge and therapy localization; a strong indicator muscle should not weaken on inspiration, either nasal or oral. If there was a blood pressure abnormality, it should be re-evaluated immediately after the cranial fault is corrected. Sometimes there is a significant reduction in the blood pressure immediately after correction; however, it may be lost before the patient returns for his next office visit. In this case, the reduction immediately after correction indicates that the right approach is being attempted but the correction is not maintained. Further evaluation of the cranium, pelvis, and other factors that influence the primary respiratory mechanism must be done so the correction is maintained.



6—25. Step 1. Contact at external occipital protuberance and glabella. Pressure at the two contacts is directed toward each other on oral or nasal respiration that did not cause a strong indicator muscle to weaken.



6—26. Step 2. Pressure is continued on the glabella and external occipital protuberance as in Step 1, and inferior motion is added to the posterior arch of atlas and the spinous processes of C2 and C3.

Rotational Cranial Faults

Rotational cranial faults are those having lack of or improper movement about the sagittal or vertical axes as their primary aspect. This, then, relates primarily to the lateral or medial movement of the temporal bone squama, rotation of the frontal bone

about the vertical axis, and rotation of the occipital and sphenoid bones about the sagittal axis. The cranial faults listed in this group are the following: temporal bulge, parietal descent, internal and external frontal, and universal.

TEMPORAL BULGE CRANIAL FAULT

The temporal bulge cranial fault is characterized by a muscle which is weak because of the fault becoming strong when inspiration is stopped in the middle and held. This is known as a "half-breath held in." A muscle weak because of the fault is very often on the side of the temporal bulge.

The half-breath held in will usually strengthen a weak muscle associated with this fault anywhere in the middle range of inspiration. In some instances, only a small range of held inspiration will cause the muscle to strengthen. To observe for this limited range, a patient completely exhales, takes in a small amount of air, and holds it while the muscle is tested. If the muscle doesn't strengthen, some additional air is inspired and the test repeated. This is continued until the exact amount of inspiration that causes the muscle to strengthen is found. This procedure is not often necessary; it is done only when there is a subclinical fault that is difficult to define.

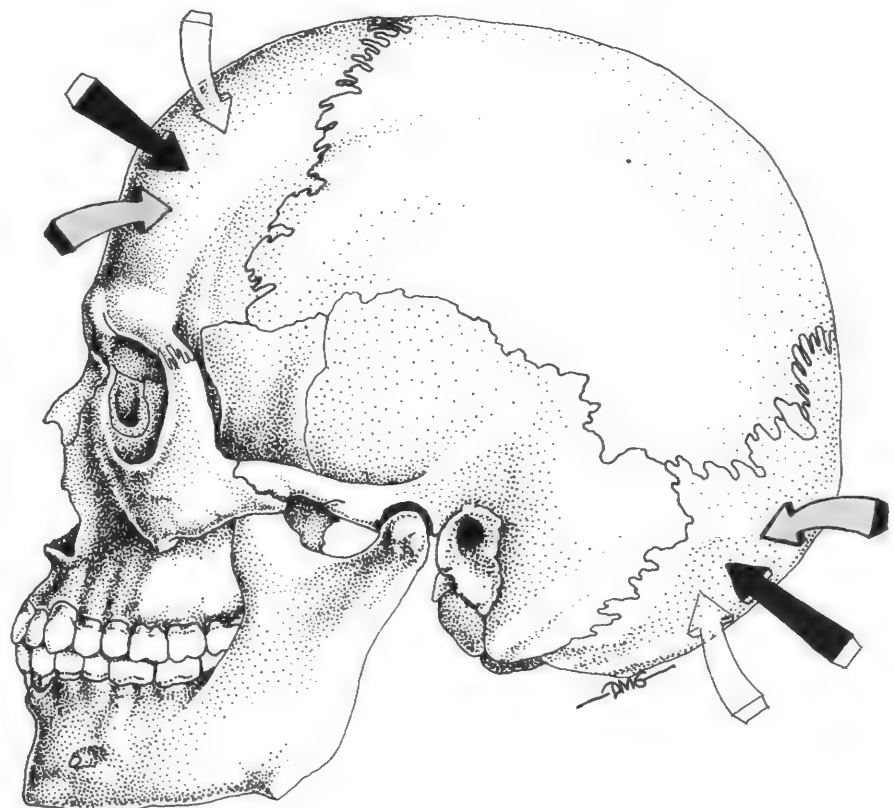
Mechanics of the Fault

The temporal bulge cranial fault is aptly described by its name. The squamous portion of the temporal bone is lateral and anterior, often causing a bulging appearance of the skull on that side. The opposite side of the skull is often medial or in the direction of a concavity. This bulge on one side and compensating medial position of the other give the appearance of a banana and has been

called a "banana head," which is synonymous with a temporal bulge cranial fault.

In addition to the flared squama, the tip of the mastoid process is posterior and medial. The position of these parts is the result of the bone having rotated around its general axis, which is through the petrous portion of the temporal bone. The superior ridge of the petrous portion is anterior and lateral.

Very often a bulge at the squamosal suture area will be observed on palpation. It may even be visually



6—27. The various arrow combinations indicate vectors of challenge for the temporal bulge cranial fault. The black arrows are challenged together for one torque into the cranium, the gray arrows for another, and the clear arrows for yet another.

observed, and there may be a flaring of the ear on that side.

Usually all aspects of an examination of the cranial primary respiratory mechanism will correlate. Since a temporal bulge is associated with an inspiration assist, it is usually present on the side of an inspiration assist cranial fault or sphenobasilar inspiration assist cranial fault. Although this correlation is usually present, there are times when the skull will have cranial faults which do not conform to the rest of the skull's activity. The final determination of how a correction is to be made is related to the way the skull challenges.

Mechanics of Correction

A temporal bulge is often corrected without direct treatment when an inspiration assist or sphenobasilar inspiration assist cranial fault is corrected with the use of accurate vectors. The latter cranial faults are considered flexion-extension faults; however, as mentioned previously, nearly all cranial faults have a three-dimensional characteristic. By using accurate vectors in making correction, the three-dimensional characteristic of the fault is taken into consideration. For this reason, the maximum challenge observed when evaluating either of the latter cranial faults is the best approach for correction. It not only corrects the flexion and extension aspects of the involvement; it also takes the rotational factor into consideration.

It may be necessary to correct a temporal bulge cranial fault because it was not accomplished with an inspiration assist or sphenobasilar inspiration assist cranial correction. If so, it is necessary to accurately challenge to determine the vector for correction.

Therapy Localization

Therapy localization will often be positive directly over the squama of the temporal bone. There will also probably be positive therapy localization when the patient places a finger in the external auditory meatus. Care must be taken that the positive therapy localization is actually indicative of a temporal bulge cranial fault, since other structures in these areas may show positive therapy localization.

Challenge

Challenge is two-handed, with pressure applied simultaneously at both locations. One contact is on the occipital bone close to the asterion; the other contact is on the frontal bone, close to the pterion. Contact is usually made with the physician's thumbs, and pressure is applied to attempt to increase the temporal bone bulge. The first vector applied is generally the thumbs pressing toward each other,

Examination and Treatment of Cranial Faults

after which a previously strong indicator muscle is tested for weakening. The vector is changed by applying pressure in a somewhat superior direction with one thumb and an inferior direction with the other, while continuing to try to increase the temporal bone bulge. This places a torque into the skull. Challenge is repeated with varying degrees of torque, with the thumbs applying torque to the skull in opposite directions and increasing the bulge. In other words, if one thumb moves superiorly, the other will move inferiorly. Rarely, if ever, will both move superiorly or inferiorly. The optimum challenge is found when maximum weakening of the indicator muscle develops.

Muscle Correlation

Associated with the temporal bulge cranial fault is bilateral pectoralis major (clavicular division) weakness. Although other muscles may be weak with the fault, the bilateral clavicular division seems to be the best muscle to use for evaluation. The muscles may test strong individually on manual muscle testing, but will be weak when tested together. The exact reason for this muscular weakness with the temporal bulge cranial fault is unknown. It is clinically observed that bilateral weakness most often indicates a temporal bulge, but it is not pathognomonic of it. When the bilateral weakness is strengthened with a half-breath inspiration, the test does become pathognomonic. Correction of the temporal bulge will return strength to the bilaterally weak muscles.

In some instances, a bilateral pectoralis major (clavicular division) weakness may be masked by a bilateral lower trapezius weakness. This is discussed in Volume I under "Fixation Masking Patterns." If the bilateral pectoralis major (clavicular division) muscles test strong but bilateral lower trapezius muscles are weak, test for and correct the dorsolumbar fixation which is nearly always present. Then re-test for the bilateral pectoralis major (clavicular division) weakness. It will nearly always appear if body language indicates a temporal bulge cranial fault.

The temporoparietalis and the auricularis anterior, superior, and posterior are often involved and usually hypertonic, requiring applied kinesiology treatment to the Golgi tendon organ or neuromuscular spindle cell to reduce the muscles' tension.^{7, 8} The muscles of mastication significantly involved with the temporal bulge cranial fault are the temporalis, masseter, and internal pterygoid. To balance the masticatory muscles, cranial faults and the temporomandibular joint (discussed in Section II of this text) must be corrected. Proprioceptive treatment to the muscles may also be needed.

Examination and Treatment of Cranial Faults

Conditions Correlating

Whenever a temporal bulge cranial fault is present, there is a probability of a parietal descent cranial fault on the opposite side; an evaluation for it should be done. This is especially true if there is an expiration assist cranial fault, or a sphenobasilar expiration assist cranial fault on the opposite side.

The temporal bulge appears to influence the vagus nerve more than any other fault. This is probably the reason there are often indications of digestive disturbances, especially hypochlorhydria. The bilateral pectoralis major (clavicular division) weakness is correlated in applied kinesiology with both the temporal bulge cranial fault and hypochlorhydria. Having the patient suck on a hydrochloric acid tablet will abolish the bilateral pectoralis major (clavicular division) weakness. Care must be taken that the use of this chemical does not mask the presence of a temporal bulge. Hydrochloric acid tablets are often given for certain types of conditions. Their use can mask the presence of a temporal bulge so that the correction that would eliminate the need for the hydrochloric acid tablet is not obtained.

Another interesting chemical aspect of this cranial fault is that a susceptible individual can appear to have no cranial faults, but chewing an antacid tablet will immediately produce clinical evidence of a temporal bulge. When repeated correction of a temporal bulge is necessary, the patient is often taking antacids. Elimination of their

use frequently allows the temporal bulge correction to be maintained and the symptoms alleviated. This is discussed more thoroughly in Volume V.

Correction

Correct by exaggerating the lesion with a two-handed contact, as described for the challenge. Pressure should be applied in the direction that caused maximum weakening of an indicator muscle during challenge. The pressure is applied during inspiration, with the maximum amount of pressure during the middle phase. If the phase of inspiration that strengthened a weak associated muscle is not precisely in the middle, the pressure should be applied at the point which caused the muscle to strengthen. The pressure should be repeated four or five times with the patient's inspiration, or until the physician feels the skull yield to the therapeutic effort. Re-evaluate with challenge, therapy localization, and testing the weak associated muscle for strengthening to determine that the correction has been obtained. Although a temporal bulge is often indicated by an apparent bulging of the lateral aspect of the skull, it is not a criterion for attempting a temporal bulge correction. The skull may have a bulge, but it may have ossified in such a way that normal motion is present with no nerve entrapment or other dysfunction. By making corrections only when indicated by challenge, iatrogenic problems are circumvented.



6—28. Pressure is exerted in the direction of optimum challenge. Maximum pressure is exerted in the half-breath phase of inspiration.

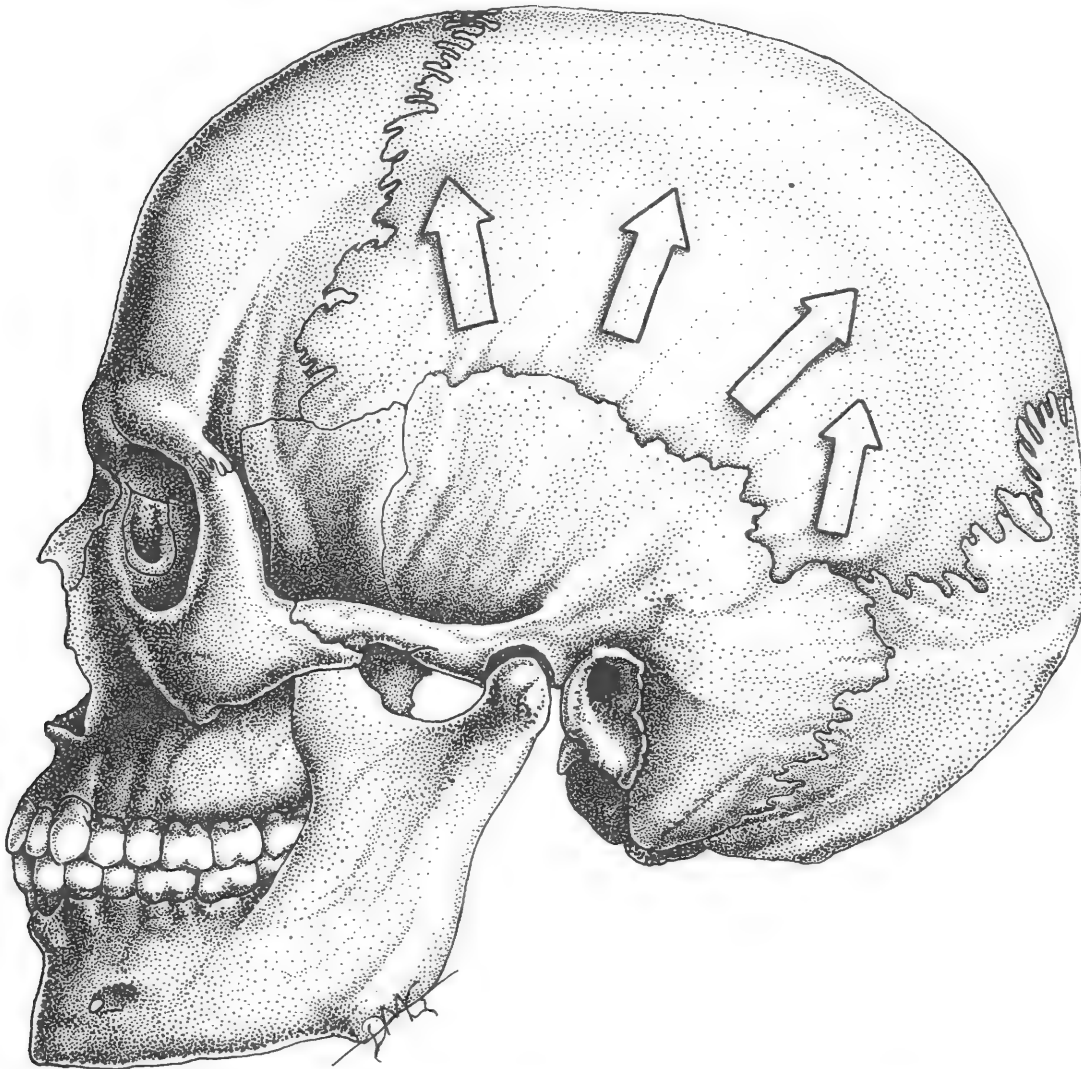
PARIETAL DESCENT CRANIAL FAULT

The parietal descent cranial fault is characterized by a weak associated muscle — usually on the side of the fault — becoming strong when the patient stops in the middle of expiration and holds that phase of respiration. As with the temporal bulge, the half-breath held out is generally in the middle of the respiratory phase. Sometimes the range of the expiration phase which strengthens the muscle can be very limited. To obtain strength in a weak associated muscle, it may be necessary to have the patient take a full inspiration and slowly exhale, stopping frequently to test the muscle suspected to be associated with the fault for strengthening. It may take many tests to find a very limited range in the expiration phase that will cause the muscle to

strengthen when the patient holds that phase. A previously strong indicator muscle may also be examined for weakening with a step-by-step inspiration test.

Mechanics of the Fault

The parietal descent cranial fault is opposite that of the temporal bulge. The squama of the temporal bone is medial and slightly posterior, while the tip of the mastoid process is anterior and lateral. As the name suggests, the parietal bone descends, causing a jamming of the squamosal and parietomastoid sutures. It appears that the major jamming is at the parietomastoid suture, as there is significant sliding capability along the squamosal suture.



6—29. Because of the primarily sliding nature of the suture involved with this cranial fault, both a separating and an approximating challenge may be positive. The correction is always made by lifting the parietal away from the temporal bone.

Examination and Treatment of Cranial Faults

Mechanics of Correction

Quite often the parietal descent cranial fault will be corrected when an expiration assist or spheno-basilar expiration assist cranial fault is corrected. In some cases, it is necessary to lift the parietal bone away from the temporal bone to allow normal motion to return. Correction of most cranial faults in applied kinesiology is done with a rebound action in the adult; in this case, the correction is a direct lifting of the parietal bone.

Therapy Localization

There will often be positive therapy localization along the squamosal and the parietomastoid sutures. Also, therapy localization will often be positive when the patient's finger is placed in the external auditory meatus. Again — as with other cranial faults — it is important to consider therapy localization only as an indication that there may be an involvement present; it is not pathognomonic of the involvement.

Challenge

Challenge for this cranial fault does not have the vectoring requirement of other cranial faults in determining the direction of correction. The fault is best diagnosed by a combination of challenge and the half-breath held out described previously. The usual challenge is to lift the temporal border of the parietal bone and observe for weakening of a previously strong indicator muscle. Sometimes a positive chal-

lenge is elicited by pushing the temporal border of the parietal bone inferiorly, jamming the parietomastoid and squamosal sutures.

Muscle Correlation

Often there is a unilateral scalene muscle group weakness when this fault is present. The muscles will strengthen with the half-breath held out. This correction is very important in thoracic outlet syndromes.

The temporalis muscle is often hypertonic with this cranial fault, requiring neuromuscular spindle cell or Golgi tendon organ treatment for relaxation. The temporoparietalis and the auricularis anterior, superior, and posterior may also be involved in a similar manner.

If positive correlation cannot be observed when a parietal descent cranial fault is suspected, have the patient clench his teeth, activating the temporalis muscle; then re-evaluate. Often a subclinical parietal descent cranial fault will then be observed. Care must be taken that it is not an involvement with the TMJ or teeth that is being evaluated rather than the parietal descent cranial fault. These can be differentiated as described in Section II of this text.

Pain Location

A temporal headache is often present with the parietal descent cranial fault, due to nerve irritation in the squamosal and parietomastoid sutures. A



6—30. Strongest lift of the parietal bone is generally done as the patient passes through one-half expiration. The sagittal suture is protected from jamming by the physician's thumbs separating it at the same time the parietal bone is lifted.

procedure was developed in osteopathy to differentiate this as a cause of headache. An effort is made to separate the sutures with digital pressure on the temporal and parietal bones, observing for relief of the headache while this pressure is held.¹⁸ Careful palpation along the squamosal suture will often reveal a ridge where the temporal bone is riding over the parietal bone. This may be difficult to feel if the temporalis muscle is hypertonic. There is often pain at the 1st rib head, probably due to scalene muscle imbalance.

Conditions Correlating

The parietal descent cranial fault is often accompanied by a temporal bulge on the opposite side; evaluation should be made for this. The parietal descent correlates with an expiration assist or sphenobasilar expiration assist cranial fault. Failure to find these correlations is most often observed if there is a combination of many faults with the accompanying complexity.

Correction

The parietal descent is corrected on expiration. The doctor's contact lifts the parietal bone away from the temporal bone. Usually a two-handed

Examination and Treatment of Cranial Faults

contact is made, with the physician's digits just superior to the squamosal and parietomastoid sutures to lift the parietal bone. An additional contact is made with the physician's thumbs on both sides of the sagittal suture to keep it separated. Correction is made as the patient exhales, with the greatest amount of pressure applied during the middle portion of the exhalation. If the phase of exhalation that strengthened an associated weak muscle differs from the middle phase of expiration, the greatest lift should be applied at that point. As the physician tractions up on the parietal bone, the thumbs — which are placed on both sides of the sagittal suture — spread the sagittal suture. This prevents jamming of the sagittal suture, which could create an iatrogenic cranial fault as a result of correcting the parietal descent. The parietal bone is lifted four or five times, or until the physician feels skull movement from the therapeutic effort.

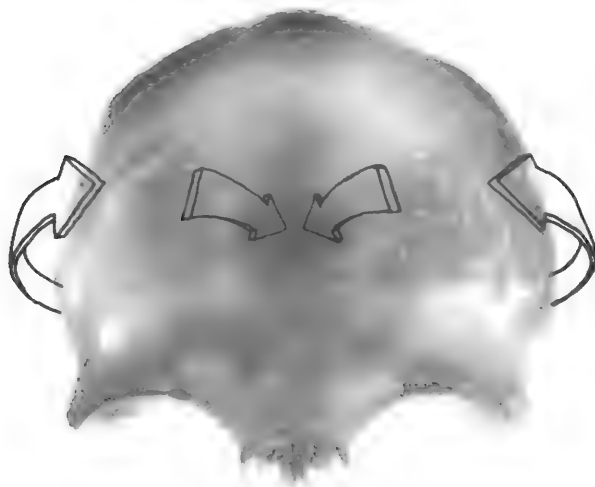
After it appears there has been correction of the cranial fault, re-evaluate with challenge, therapy localization, and testing the weak associated muscle for strengthening to determine that correction was actually obtained. There should also be a significant reduction of the temporal headache if it was present, and strengthened medial neck flexors.

Examination and Treatment of Cranial Faults

INTERNAL FRONTAL CRANIAL FAULT

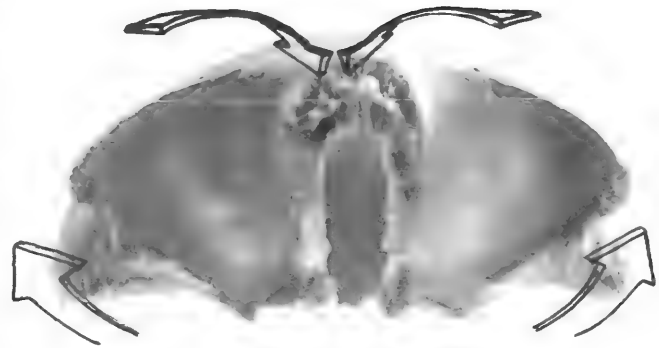
The term “internal frontal” cranial fault infers that the frontal bone is primarily involved. Actually, many bones are usually involved. The fault relates to the frontal bone as if the metopic suture were persistent throughout life, thus making a right and a left frontal bone. In the adult, there is limited motion of the frontal bone; in most cases it is limited to the flexibility of living bone or rotation of the total bone, which is minimal. Frontal faults are primarily involved with the sphenoid, ethmoid, and facial bones — including the palatine, maxillary, vomer, zygomatic, and other smaller bones.

The term “internal frontal” refers to rotation of the frontal bone about the vertical axis. In applied kinesiology, the term “internal” refers to the median portion of the anterior aspect of the frontal bone, meaning that the metopic suture area moves internally or posteriorly. This is confusing, because DeJarnette’s sacro occipital technique^{1,2} and cranial osteopathy¹¹ refer to the lateral aspect of the



6—31. *Internal frontal bone rotation as defined in applied kinesiology.*

squamous portion of frontal bone movement. With their terminology, an internal frontal refers to the lateral aspect of the squamous portion of the frontal bone moving medially, which would mean an external movement of the metopic suture area. Thus in these two systems, an internal frontal would be exactly opposite that referred to in applied kinesiology. In this text, the applied kinesiology terminology of motion at the metopic suture area will be maintained to help avoid confusion among those who have previously studied AK. When there is a reference to internal or external rotation of the frontal bone, the structure involved will be mentioned if it differs from the metopic suture area.



6—32. *Inferior view of internal frontal bone rotation.*

Mechanics of the Fault

The frontal bone, considered as two lateral halves, has two axes of rotation. For each half, the axis generally goes through the orbital surface off-centered anteriorly and medially to travel vertically through the frontal eminence. Internal rotation — referring to the metopic suture area — moves that structure posteriorly, while the lateral aspect of the squamous portion (lateral angle) moves anteriorly and laterally. The ethmoid notch, which contains the crista galli and cribriform plate of the ethmoid, widens.

The sphenoid rotates about its sagittal axis, with the internal frontal side inferior on the side of internal frontal rotation. There may also be some misalignment of the sphenoid in the flexion and extension aspects of the sphenobasilar junction, as described earlier.

The bones of the orbit are generally distorted, usually revealing a retruded and smaller-appearing eye on the side of internal frontal rotation.

The intervening bones between the frontal and the sphenoid — which include the palatine, maxilla, vomer, and ethmoid — are rotated and flexed or extended to accommodate the misalignments of the frontal and sphenoid.

Mechanics of Correction

Correction of the internal frontal cranial fault is done in three steps. The first step is pressure applied at the cruciate suture, the second on the pterygoid process of the side of involvement, and the third on the pterygoid process opposite the side of involvement. Because there are several contacts, correction is probably obtained by a combination of increasing the cranial fault to unlock it so that the skull may rebound into normal function, and by direct correction.

Examination and Treatment of Cranial Faults

Pressure applied at the cruciate suture is transmitted through the vomer to the sphenoid and ethmoid, and through the maxilla to the frontal, sphenoid, zygomatic, lacrimal, and nasal bones. This pressure also influences the palatine bone, which transmits its force to the sphenoid by way of the pyramidal process to the pterygoid process, and by its sphenoidal process to the anterior portion of the body of the sphenoid. The palatine also influences the orbit by its orbital process.

Therapeutic force applied to the pterygoid processes directly influences the sphenoid and indirectly influences several other bones, including the palatine, frontal, ethmoid, and temporal. As the physician gains experience in applying pressure to the pterygoid processes, an ability to feel the structure move should develop.

Therapy Localization

Positive therapy localization for a frontal cranial fault has been considered to be over the maxilla just under the zygomatic bone, or on the anterior aspect of the frontal bone above the orbit. This therapy localization leaves a lot to be desired because of other factors in those areas that may therapy localize, such as stress receptors, individual suture involvement, sinusitis, etc. A better therapy localization can be obtained by having the patient place a finger intra-orally over the pterygoid and pyramidal processes on the side of apparent involvement. Although this is an improved therapy localization, it does not specifically indicate a frontal bone cranial fault, or whether the involvement is internal or external rotation. Therapy localization for any cranial fault should be used only as a guide to indicate the possibility of a cranial fault. Re-test after a therapeutic attempt to determine if the positive therapy localization has been eliminated.

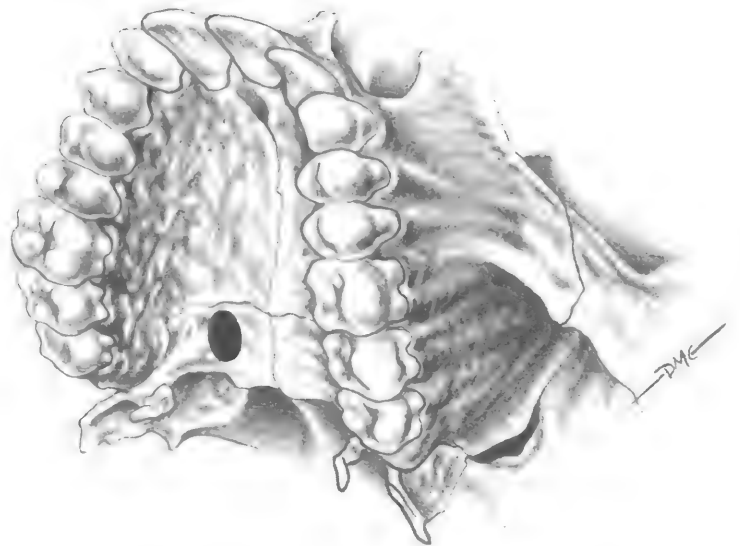
Challenge

Challenge is the primary method for determining the presence of an internal frontal cranial fault. A rebound pressure is applied to the malar surface of the zygomatic bone, primarily in a medial, slightly posterior direction. In the presence of a positive challenge, a previously strong indicator muscle will weaken. If the neck flexors are strong in the clear, they are the best muscles to use as indicators; however, any muscle strong in the clear may be used. It is sometimes necessary to vary the vector of force on the zygomatic bone to obtain a positive response.

Challenge in the usual sense to determine a treatment vector does not appear to be applicable in the frontal cranial faults. There is a modified type of



6—33. Challenge to malar surface of zygomatic bone is best method for determining whether an internal frontal fault is present.



6—34. Various vectors of force are applied in the general area of the lateral palatamaxillary suture to find the direction that relieves eye pain upon digital pressure to the maximum amount.

challenge regarding eye tenderness that is helpful in determining the exact vector for the first step of internal frontal cranial fault correction.

There will nearly always be tenderness of one or both eyeballs upon digital pressure. The examiner presses on a closed eyelid to have the patient determine the level of discomfort. If there is no discomfort when pressing posteriorly on the eye, evaluate for discomfort in the upper, lower, medial, and lateral borders of the eye. If the examiner still cannot find any eye discomfort, have the patient press on the eye to locate the area of discomfort. (Take care that the patient is not wearing contact lenses before applying the pressure.) After the patient has determined the pain level of the more tender eye, the physician applies digital pressure to the cruciate suture area. The pressure is approximately three or four pounds, in a generally cephalad

Examination and Treatment of Cranial Faults

direction. With the pressure applied, the physician again presses on the closed eyelid with as close to the previous pressure and location as possible to determine if the discomfort has decreased. If not, the vector of pressure at the cruciate suture area is then changed somewhat to include slightly anterior, posterior, lateral, or medial pressure, and the eye is re-evaluated for tenderness. When the optimum vector for corrective pressure is found, tenderness of the eye will be dramatically reduced or eliminated. In most instances, it is easy to find this vector because it is generally in a superior direction at the lateral palatomaxillary suture, close to the molar teeth. The added vector, in addition to being superior, can be any direction in the 360° arch. It may be almost entirely lateral, lateral-anterior, lateral-posterior, or even medial in some cases. In a few cases, finding the correct vector may be quite difficult and require several efforts. It may also be necessary to contact the palate more anteriorly or posteriorly. The point and vector combination which causes the greatest diminishment of eye pain may be on the palatine bone or more anterior on the maxilla. The major concern is to find the location and vector on the palate which reduces the pain in the patient's eye, as evaluated by digital pressure.

The vector of pressure on the palate which reduces or eliminates the eye pain will be the vector for the first step in correction. This pressure is transmitted to the sphenoid bone by way of the vomer and the palatine bones. It appears that pain reduction in the eye is a result of relieving strain in the bony orbit as the bones are moved into a more normal position. For the mechanics of motion in the closed kinematic chain of the skull, see pages 79-86.

Muscle Correlation

The muscles most often weak as a result of this cranial fault are the neck flexors, which include the medial neck flexors and the sternocleidomastoid. These are often weak unilaterally or bilaterally. They will sometimes strengthen with certain phases of respiration, such as a full breath held in; however, respiratory strengthening of the neck flexor muscles is not consistent and cannot be used as an indicator for this cranial fault. After the cranial fault has been corrected, the muscles will be strong if they were weak as a result of the involvement.

There are many intrinsic skull muscles which may be associated with this cranial fault. The occipitofrontalis may be involved; if so, it is usually hypertonic. Treatment to the proprioceptors to relax the muscle may be required in either the occipital or the frontal bellies. The procerus and corrugator supercilii are often hypertonic and require proprio-

ceptive treatment. These muscles are often involved when there is a restriction of nasal breathing or interference with proper ionization.^{7,8} (For a complete discussion of ionization, refer to Volume III.) Body language for contraction of the procerus and corrugator supercilii is persistent wrinkles and furrowing above the nose and between the eyebrows.

Any of the muscles of the eyelids, and some muscles of the nose, can be involved. Again, the muscle is generally hypertonic; treatment to the proprioceptors is necessary to return normal tonicity.

Muscles of the mouth — especially the zygomatic major and minor — may be involved. They may be weak, but more often they are hypertonic.

The hyoid muscles and the muscles of mastication and the temporomandibular joint are often involved. The internal and external pterygoid muscles, having their origins on the pterygoid processes, are quite frequently imbalanced. The imbalance generally causes a torque in the sphenoid about its sagittal axis. Evaluation and treatment of the TMJ and muscles of mastication should routinely be done when there is a frontal cranial fault. This is discussed in Sections II and III of this text.

Pain Location

Tenderness of the eyeball on digital pressure has already been mentioned. As stated above, the patient will often complain of a headache "behind the eye." The pterygoid processes are usually quite tender, and there is tenderness around the greater wing of the sphenoid, generally bilaterally. It tends to be somewhat greater on the side of internal frontal rotation. There may also be pain on digital pressure over the supraorbital notch on the side of internal rotation.

Conditions Correlating

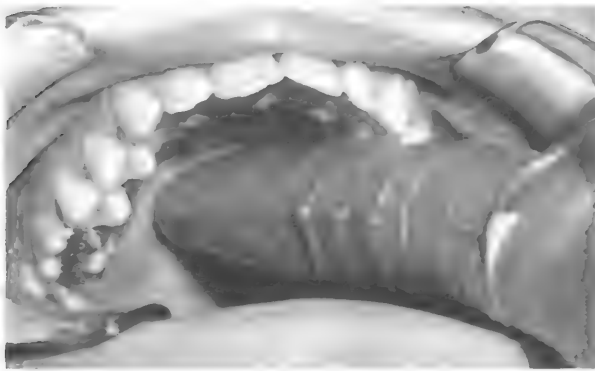
The patient with a frontal cranial fault will often complain dramatically of a headache that centers "behind the eye." The description may be, "It seems like someone is trying to pull my eye out," or "It feels like a sharp, hot poker is being jammed into my eye." This pain will often be immediately eliminated when the condition is corrected. There is frequently an involvement with visual disturbances, such as visual acuity or the eyes not working together properly. Sinusitis, rhinitis, or difficulty in nasal breathing is also associated with this cranial fault. In this case, evaluation for active neurolymphatic reflexes and retrograde lymphatic should be done.

Body language generally consists of a larger nares and smaller orbit on the side of internal frontal rotation at the metopic suture.

Correction

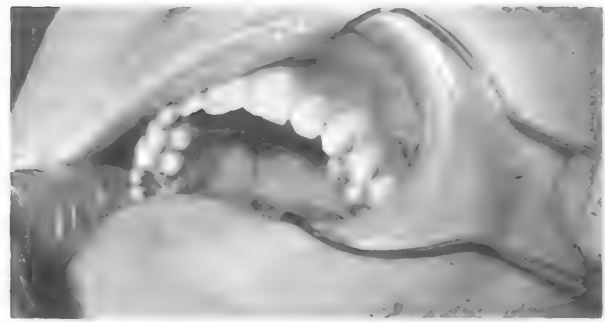
Correction for an internal frontal cranial fault is accomplished in three steps.

Step 1. Locate the vector of pressure that removes the tenderness at the eye on digital pressure. This pressure — three to four pounds — should be held for twenty to forty seconds. Occasionally, the physician can feel the skull yield under his pressure; however, this is not a consistent observation. It improves with the physician's experience. If the patient has no eye tenderness on digital pressure, the pressure is held at the cruciate suture near the second molar in a directly superior direction on the side of internal rotation.

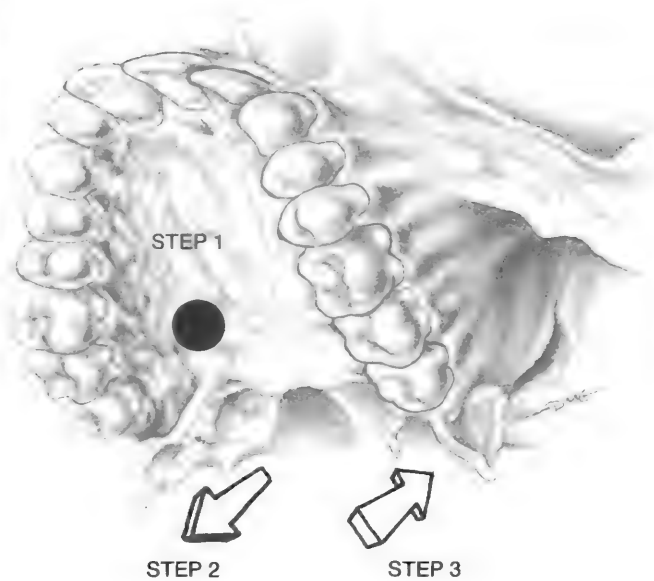


6—35. Illustrations are for a right internal frontal fault. Step 1. Hold pressure in direction that relieved eye pain.

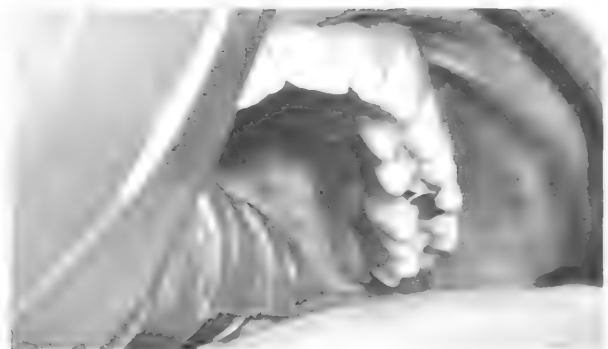
Step 2. Contact the pterygoid process on the side of internal rotation. The approach to the pterygoid process is usually best accomplished on the buccal side. The examiner slides his finger between the teeth and cheek to the pterygoid process, hooking above the lateral pterygoid plate to pull caudally on the pterygoid process. This pressure is held for ten to twenty seconds. The physician will usually be able to feel the sphenoid move inferiorly after pressure is held for a time. When the physician is adept in feeling this movement, it is the best indicator for determining how long to hold the pressure. It may be necessary to have the patient open or close his mouth slightly to move the mandible, giving access to the lateral pterygoid plate. In some instances, it is easier to contact the pterygoid process on the lingual side because the entire pterygoid process is medial. In this case, the finger is hooked above the medial pterygoid plate and a caudal pressure is applied. Sometimes it is best to use a two-finger contact, with one finger approaching from the buccal side and the other from the lingual side.



6—36. Step 2. Buccal approach to lateral pterygoid plate. It may sometimes be better to contact the medial plate with a lingual approach.



6—37.



6—38. Step 3. Lingual approach to inferior pterygoid plates.

Examination and Treatment of Cranial Faults

Step 3. Contact the pterygoid process on the side opposite internal frontal rotation. This contact is made on the distal aspect of the pterygoid plates, and pressure is applied in a superior, slightly posterior direction. Here again the pressure is held for ten to twenty seconds, or until the physician feels the sphenoid move.

The pterygoid processes are generally very tender. The physician's technique must take into consideration the amount of pain caused to the patient, using just enough pressure to accomplish the correction. This is generally approximately three to four pounds. Excessive pressure can fracture a pterygoid plate. Although there have been no reports of permanent problems developing from fracture of

a pterygoid plate, it is obvious that it should be avoided. When the pterygoid plate is fractured, swelling and a sore throat will follow.

There is no specific breathing pattern for the patient during correction of the internal frontal cranial fault. It seems best to have him breathe normally during the process.

After the therapeutic effort, the patient should be re-evaluated using challenge, therapy localization, and testing of associated weak muscles for strengthening. There will nearly always be a significant reduction or elimination of the pain at the eye on digital pressure. This is a good indicator of the effectiveness of the correction. If correction was not obtained, the therapeutic effort should be repeated.

EXTERNAL FRONTAL CRANIAL FAULT

This cranial fault refers to rotation of the frontal bone around the same axis as that described for the internal frontal cranial fault. In this fault, the metopic suture area of the frontal bone moves anteriorly, while the lateral border of the frontal squama (lateral angle) moves medially and posteriorly. The ethmoid notch narrows slightly.

Mechanics of the Fault

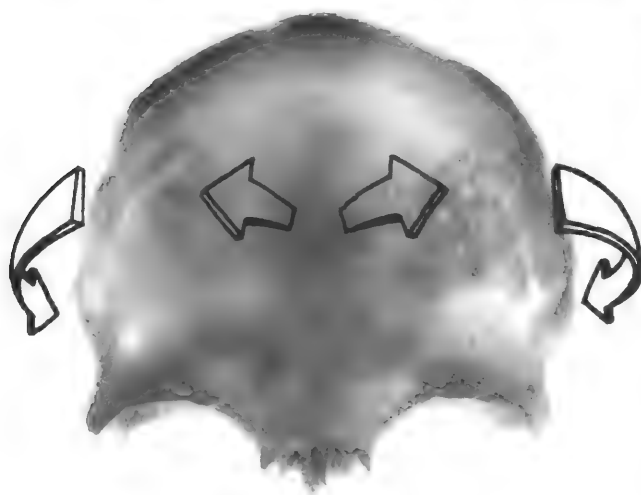
Like the internal frontal cranial fault, there is limited motion of the frontal bone with the external frontal cranial fault, and considerable involvement of other bones such as the sphenoid, ethmoid, palatine, maxilla, vomer, and other bones of the eye orbit.

The sphenoid is superior on the side of the

external frontal cranial fault. Its misalignment, along with other bones, causes the orbit to be distorted. It usually gives the appearance of a protruded, enlarged eye on the involved side.



6—40. Inferior view of external frontal bone rotation.



6—39. External frontal bone rotation as defined in applied kinesiology.

Mechanics of Correction

Correction is obtained on a rebound basis, with forces applied to bones other than the frontal. The correction is done in two steps.

Step 1. Pressure is applied to the palate in an area generally slightly posterior to the lateral aspect of the cruciate suture on the side opposite the external frontal. This force influences the sphenoid through the palatine bone and its articulation with the pterygoid process and the anterior portion of the sphenoidal body. The force to the palate is transmitted to the sphenoid and ethmoid by way of the vomer. The maxillary bone also places direct pressure on the frontal with its frontal process, and indirect pressure through the zygomatic bone.

Step 2. Contact the pterygoid process on the side of external frontal rotation. This directly influences the sphenoid.

There is no specific respiratory correlation with this cranial fault. It will often be corrected secondarily when inspiration and/or expiration assist cranial faults are corrected, and also with the sphenobasilar cranial faults.

Therapy Localization

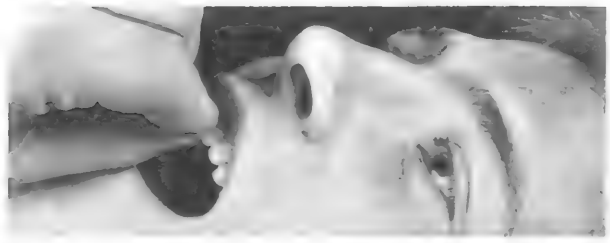
Optimum therapy localization for this cranial fault is the same as for an internal frontal fault. Previously it had been considered to be over the maxilla — just under the zygomatic bone — or on the frontal bone above the orbit. Improved therapy localization can be obtained when a patient places his finger on the pterygoid process on the side of involvement. No therapy localization is pathognomonic of this cranial fault; it simply indicates that it may be present. There are other factors which may cause positive therapy localization in this area. Therapy localization is a valuable indicator, after therapeutic attempts have been made, in determining if correction has been obtained.

Challenge

Challenge is the positive indicator for an external frontal cranial fault. It is accomplished by pulling caudally on the central incisor on the side being evaluated. A positive challenge is indicated when a previously strong indicator muscle weakens after the traction on the central incisor. This challenge does not provide information on how to correct the cranial fault, as it does with many other faults.

Some in applied kinesiology challenge for an external frontal cranial fault by placing an inferior vector of force into the maxilla, over the skin covering the central incisor. This challenge is sometimes positive when there is an external frontal cranial fault, but it is not as effective for locating the fault as traction on the tooth.

A modified type of challenge to determine the treatment vector is the same as that used for the internal frontal cranial fault. There will typically be eye tenderness on digital pressure. After evaluation for eye discomfort, as done with the internal frontal cranial fault, the physician places pressure at the first treatment point on the hard palate, usually slightly posterior to the cruciate suture on the opposite side of external frontal rotation. Applying various vectors of force in a generally superior direction, the eye is re-tested for tenderness until the vector is found which greatly diminishes or eliminates the eye tenderness. This will be the direction for the therapeutic pressure in the first step of correction.



6—41. Patient or examiner places an inferior challenge on the central incisor and releases it; then an indicator muscle is tested for weakening.

Muscle Correlation

The neck flexors — including the medial flexors and the sternocleidomastoid — are often weak in association with this cranial fault. Using respiratory assist to strengthen the weak muscles is not a good method to determine if the cranial fault is present. The muscles may or may not strengthen with a phase of respiration indicating cranial association. This is unusual in reference to a muscle which is weak as a result of a cranial fault.

As with the internal frontal cranial fault, the occipitofrontalis and muscles of the eye and mouth may be involved. They are usually hypertonic, and require proprioceptive treatment for relaxation.

The muscles of mastication are often involved, especially the internal and external pterygoid muscles. Attention to the balance of all muscles of mastication and hyoid muscles is important in obtaining lasting correction of frontal cranial faults.

Pain Location

The supraciliary arch is often painful on the side of external frontal rotation. The zygomatic bone sutures are often tender on digital pressure, and the patient may complain of facial pain in that area. The eyeballs are frequently painful, usually more so on the side of external rotation.

Conditions Correlating

The orbit will appear wider on the side of external frontal rotation. As mentioned, the neck flexors will often be weak and may not necessarily regain strength with a phase of respiration. If the neck flexors are weak as a result of a frontal cranial fault, they will strengthen after successful correction of the involvement.

Sinusitis, rhinitis, or difficulty in nasal breathing is often associated with frontal cranial faults. After correction of the cranial fault, it may be necessary to use prolonged neurolymphatic reflex treatment for the

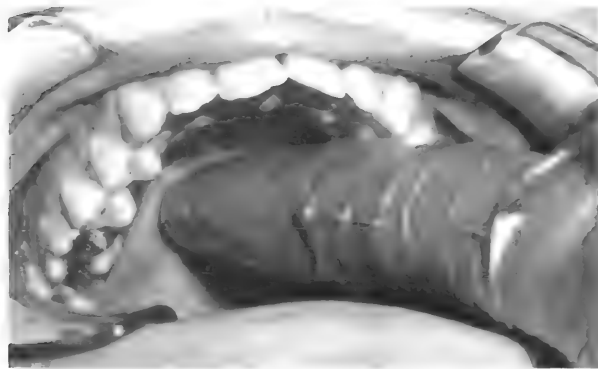
Examination and Treatment of Cranial Faults

neck flexors to drain the area most efficiently. Retrograde lymphatic treatment may also be needed to improve general lymphatic drainage of the body. (Sinus conditions are discussed more thoroughly in Volume V.)

Correction

Correction for an external frontal cranial fault is made in two steps.

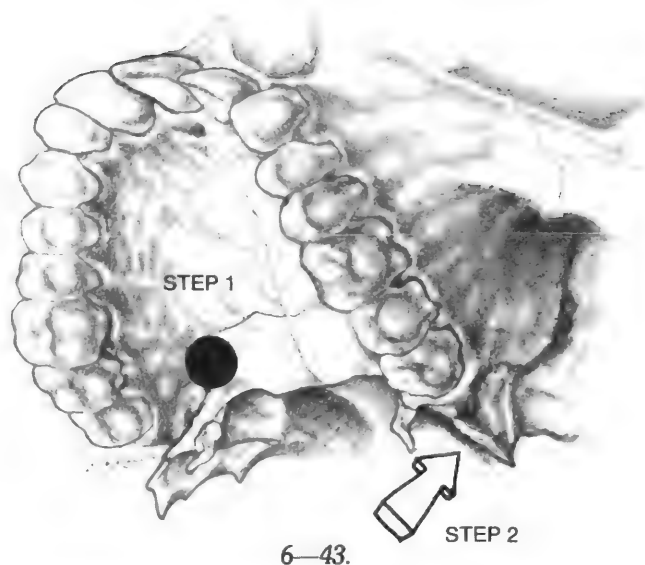
Step 1. Pressure is applied on the hard palate, usually just posterior to the cruciate suture on the **side opposite** external frontal rotation. Three to four pounds of pressure are applied in the direction previously indicated by the modified challenge, which determined what direction brings pain relief upon digital pressure on the eye. A slight movement of the hard palate is often felt by the practitioner who has trained himself in the subtle movement of the cranium. If no movement is felt, the pressure is held for twenty to forty seconds; then the second step is done before evaluating for correction.



6—42. Illustrations are for a left external frontal fault. Step 1. Apply pressure that relieved eye pain.

The pressure applied to the hard palate transmits force to the sphenoid bone through the palatine attachment to the pterygoid process and to the anterior body of the sphenoid. The pressure also influences the sphenoid and ethmoid by way of the vomer. The maxillary movement transmits force to the frontal bone through the frontal process and zygomatic bone. Because the force influences many bones, it is very important to determine the exact vector to use by the modified challenge. Each cranial fault will present a different combination of factors.

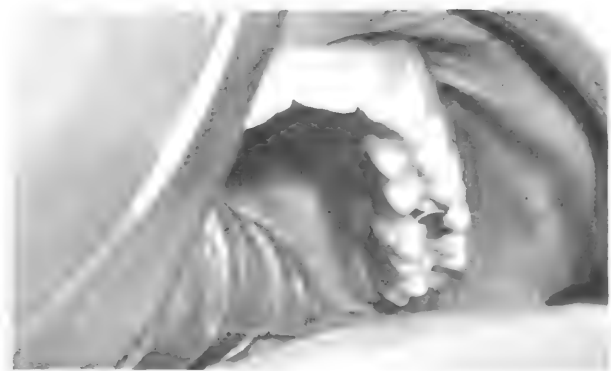
Step 2. The examiner contacts the pterygoid process on the side of external frontal bone rotation and applies a superior force of approximately three to four pounds. Again, this is held until the physician



6—43.

feels the structure yield to the pressure, or for ten to twenty seconds. As with contact of the pterygoid process for internal frontal bone correction, gentle pressure should be used and care should be taken to avoid fracturing the structure. There is not as much likelihood of an iatrogenic problem as a result of pushing superiorly on the pterygoid process as there is from pulling it inferiorly, as in the second step of the internal frontal correction. This procedure is usually not as uncomfortable to the patient as the second step of the internal frontal correction.

After the therapeutic attempt, re-evaluation should be done with therapy localization, challenge on the central incisor, and testing weak muscles associated with the fault for strengthening. Also re-evaluate the eye for pain on digital pressure. It should be greatly reduced or eliminated. If the therapeutic effort was unsuccessful, repeat the procedure. It may be necessary to hold the pressures longer, especially with chronic cranial involvements where the skull is rigid.



6—44. Step 2. Pressure applied to the inferior pterygoid plates.

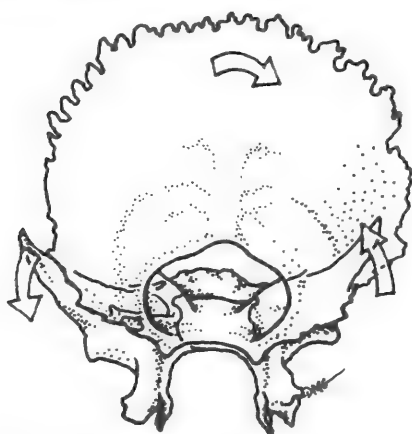
UNIVERSAL CRANIAL FAULT

The universal cranial fault is characterized by a previously strong indicator muscle weakening when a patient breathes through only one nostril. This evaluation is accomplished by blocking the nose on one side and having the patient take a deep inspiration through the open side. While the patient holds this inspiration, the examiner tests a previously strong indicator muscle to determine if it weakens. If so, a universal cranial fault is probably present. Challenge is used to differentiate this fault from an ionization problem,²⁰ which is discussed in Volume III.

A muscle weak as a result of a universal cranial fault will strengthen when a patient breathes through one nostril and holds the inspiration while the muscle is tested. The weak associated muscle will strengthen with inspiration taken through the same nostril which caused a previously strong indicator muscle to weaken.

Mechanics of the Fault

The universal cranial fault relates to counter-rotation of the sphenoid and occipital bones at the sphenobasilar junction about the sagittal axis. When this occurs, there will be associated rotation of the temporal bones on both sides.



6—45. In the universal cranial fault there is counter-rotation of the sphenoid and occipital bones about the sagittal axis.

The universal cranial fault was so named because when Goodheart first worked with this condition in applied kinesiology, he found a very high percentage of individuals with the involvement.⁶ If individuals with cranial faults are evaluated for all faults before any therapeutic efforts are attempted, a very high percentage will have the universal cranial fault. After noting that there is a universal cranial fault, the patient is evaluated for inspiration and expiration

assist by accurate vectors of challenge. After correction, it will often be found that many universal cranial faults were corrected along with the inspiration or expiration assist faults. This is also true of many other cranial faults which may be present in an untreated individual. This is why it is best to begin all cranial therapeutics with inspiration and expiration assist evaluations and corrections.

Along with the counter-rotation of the sphenoid and occipital bones in the sagittal plane, there is usually a side-bending of the two bones in the sagittal plane. This misalignment is also influenced by the inspiration and expiration assist cranial corrections.

Mechanics of Correction

It appears that the universal cranial fault correction is a direct cranial correction, as opposed to the rebound approach used with most cranial faults. The patient is prone on the adjustment table, with his face in a split headpiece. The physician contacts the occipital bone and mastoid processes of the two temporal bones to impart rotation about the sagittal axis.

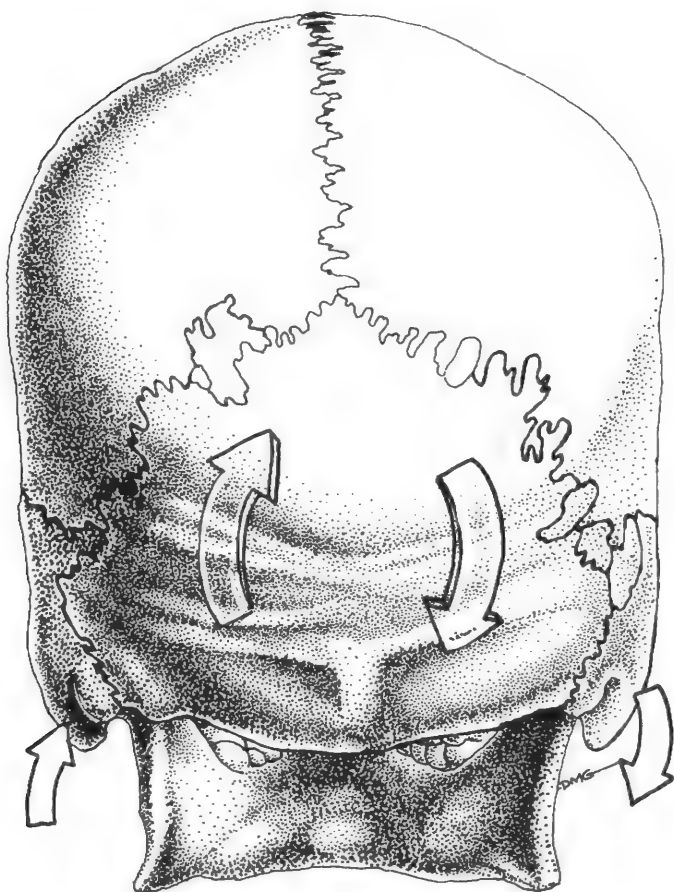
Therapy Localization

Therapy localization will generally be positive in several areas. The original description was of both hands being placed over one side of the occipital bone, not touching the parietals. There will usually be positive therapy localization along the lambdoidal and occipitomastoid sutures, too, as well as at the asterion. Again, therapy localization does not specifically indicate the cranial fault, but it does suggest that it might be there. The breathing pattern and challenge should be used to definitely determine whether a universal cranial fault is present.

Challenge

With the patient prone, one mastoid is pressed in a caudal direction and the other in a cephalad or superior direction. This is accomplished with the thumb and index finger of one hand. The other hand contacts the occipital bone, challenging it in the same direction. A positive challenge will cause a previously strong indicator muscle to weaken, or a previously weak muscle associated with the cranial fault to strengthen. Challenge is repeated in the opposite direction. The direction of challenge can be considered as either clockwise or counter-clockwise; only one direction should show positive challenge.

Correction of this cranial fault will be obtained by treatment which is opposite that indicated by the challenge of other cranial faults. In this case,



6—46. Pressure for challenge or treatment is applied on the occiput and mastoid process in the direction of the arrows, and then opposite the arrows. Contact on the occiput is kept low so as not to contact the parietal bones.

treatment will be in the direction causing a previously weak muscle associated with the cranial fault to strengthen, or opposite the direction causing a previously strong indicator muscle to weaken. This appears to correlate with this cranial fault being corrected directly, rather than by a rebound action.

Muscles Correlating

The upper trapezius and sternocleidomastoid muscles are often involved with this cranial fault. The physician often does not recognize the importance of the sternocleidomastoid muscle having a major insertion on the occipital bone, as well as on the mastoid process.

Intrinsic skull muscles often involved with the universal cranial fault are the occipitofrontalis and auricularis anterior, superior, and posterior. The occipitofrontalis can be involved with either the occipital or the frontal bellies. The auricularis group is involved because of its ability to tense the fascia of the skull. Usually the muscular involvement is hypertonicity, and treatment to the proprioceptors to relax the muscle is necessary.

Pain Location

Pain is often found on digital pressure along the lambdoidal or occipitomastoid sutures. There will sometimes be an associated suboccipital headache.

Conditions Correlating

This cranial fault is primarily associated with the occipital, sphenoid, and temporal bones. Rotation of the temporal bones — especially if there is internal rotation on one side and external on the other — appears to significantly influence the labyrinthine reflexes. Sphenoid misalignment often influences vision, possibly affecting the visual righting reflexes. A misaligned occiput may change the relationship of the condyles as they articulate with the atlas, thus influencing the head-on-neck reflexes. Organization of all these reflexes is necessary for total body organization. It has been clinically observed that persistent upper cervical subluxations and general spinal distortions are associated with the universal cranial fault. After correcting a universal cranial fault, the spine may change its character significantly from what was observed prior to the correction. It is thus

Examination and Treatment of Cranial Faults

necessary to provide close monitoring — and possible correction — of the spinal column after correcting a universal cranial fault.

On a clinical basis, the universal cranial fault is often found in conjunction with a closed ileocecal valve syndrome. This condition is discussed in Volume V.

A sacral respiratory fault will often be present with the universal cranial fault. This can be challenged by spreading the two sacroiliacs apart with lateral pressure on the posterior superior iliac spines, and then testing a previously strong indicator muscle for weakening. This fault usually corrects itself when the universal cranial fault is corrected. Occasionally it is necessary to thrust the posterior superior iliac spines away from the sacrum bilaterally. (The pelvis and its correlation to cranial faults is discussed more thoroughly in Chapter 7.)

Atlanto-occipital countertorque is also frequently present with this cranial fault. It should be routinely examined for and corrected, if necessary. Its evaluation and correction are discussed on page 219.

Correction

The contact for correction of the universal cranial fault is on both mastoid processes and on the occipital bone. The patient is prone. The physician contacts the superior aspect of one mastoid process

with either the thumb or index finger, and contacts the inferior aspect of the mastoid process on the opposite side with the thumb or forefinger; thus the thumb will be on one mastoid process and the index finger on the other. The physician's other hand makes a general, broad contact on the occipital bone, avoiding contact with the parietal bones, to impart the same torque to the occipital bone as that of the temporal bones. Contact and direction of torque are determined by challenge. Correction is in the direction of challenge that caused a muscle weak as a result of the cranial fault to strengthen, or opposite the direction that caused a previously strong indicator muscle to weaken. The torque is imparted to the skull with patient inspiration. Motion of the skull will be felt by the physician when correction has been obtained. If this ability has not been developed, repeat with four or five inspirations. In any case, re-evaluate to determine if correction has been obtained.

The split headpiece of the adjustment table basically holds the skull while correction is being obtained. Because of the pressure the headpiece applies to the facial bones, it is necessary to evaluate for the accidental creation of a cranial fault. This is usually not a problem if excessive pressure is avoided in obtaining the correction.



6—47. Universal fault correction. The physician's left hand is contacting only on the occiput.

Sutural Cranial Faults

Sutural cranial faults are limited to specific sutures which are either jammed or separated; the primary ones are the sagittal, squamosal, lambdoidal, and zygomatic sutures. The same basic principles can be applied to other sutures, such as the coronal, intermaxillary, interpalatine, etc., but these are usually corrected with other cranial faults.

The procedure of examination and correction is the same for all sutural cranial faults, with the exception of the sagittal suture. It is usually best to approach these particular cranial faults after the other, more complex, faults have been corrected; their correction often corrects the sutural faults.

SAGITTAL SUTURE CRANIAL FAULT

The sagittal suture cranial fault is usually accompanied by weak abdominal muscles. The reason for this association is unknown, but it is often clinically found.

Mechanics of the Fault

The mechanical distortion found in a sagittal suture cranial fault is that of the suture jamming. It is rarely — if ever — found separated.

Mechanics of Correction

Correction of the fault is obtained by separating the suture. It is not necessary to have a respiratory correlation with its correction.

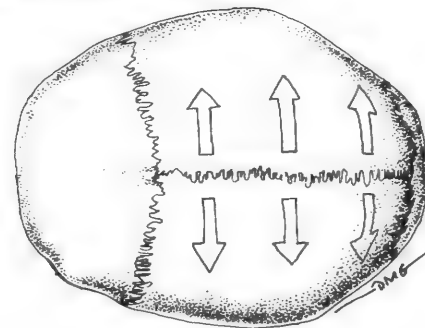
Therapy Localization

Therapy localization over the sagittal suture will cause a previously strong indicator muscle to weaken, or cause weak abdominals associated with this fault to strengthen. In the rare case where the abdominal muscles are strong in the presence of this fault, they will probably weaken upon therapy localization to the sagittal suture.

Sometimes the entire length of the suture is not jammed. The exact area of sutural involvement can be determined by therapy localization.

Challenge

It is not necessary to challenge to determine how to correct this cranial fault, since the involvement is always that of jamming. An indicator muscle will weaken if a challenge is done to approximate the parietal bones at the sagittal suture. The fault's presence is most often determined by therapy localization and associated abdominal weakness, which should be confirmed by challenge. The abdominal weakness can be related to sagittal suture jamming by observing weak abdominal muscles



6—48. Contact points for separation of sagittal suture. Care must be taken that contact is over area of involvement.

strengthening on therapy localization to the sagittal suture. As observed above, if the abdominal muscles are strong in the presence of the fault, therapy localization to the fault will generally cause them to weaken; however, it will not necessarily weaken other muscles considered as indicators.

Muscle Correlation

Any muscle that tightens the superficial and deep fascia of the epicranium can be involved with the sagittal suture cranial fault. This may include intrinsic muscles of the skull, or dysfunction remote from the skull (see Chapter 4 on the cranial muscles and fascia). When there is a recurrence of a sagittal suture cranial fault, the muscles and fascia should be evaluated. Treatment remote from the skull may be necessary; it could involve many different therapeutic approaches used in applied kinesiology.

Pain Location

There is typically pain along the sagittal suture on digital pressure. It may be severe enough to cause a spontaneous headache which is either persistent or recurrent.

Conditions Correlating

It is possible that the abdominal muscle weakness correlates with a sagittal suture cranial fault because abdominal ptosis causes a generalized traction on the fascia, which ultimately results in tightening of the superficial and deep fascia of the epicranium. This could be a vicious circle of cranial etiology which disturbs the function in such a manner that the cranial faults cause additional muscle weakness. Another observation is the important role the abdominal muscles play in pelvic support, especially in category I pelvic faults. The category I pelvic fault is especially important in the cranial-sacral primary respiratory mechanism (see page 211).

Correction

Correction is obtained with digital contact on both sides of the sagittal suture; pressure is applied for separation. There is no respiratory correlation considered with this fault; however, it appears to improve correction somewhat if the patient inspires at the time of separation.

If the suture is not jammed over its entire length, care must be taken to spread the area actually involved. As noted, this is determined by positive therapy localization. A frequent error in the correction of the sagittal suture cranial fault is failure to contact far enough posteriorly on the skull. In some instances, the physician's contact is actually over the



6—49. Sagittal suture separation.

frontal bone, failing to even contact the parietals. Therapy localization also helps eliminate this error.

After correction, the abdominal muscles should be tested for strengthening, if they were weak in association with the fault. Challenge to approximate the suture and therapy localization should also be done.

SQUAMOSAL SUTURE CRANIAL FAULT

The squamosal cranial fault is either a jamming or a separation of the squamosal suture. Most often it is jammed, and is corrected on a rebound basis. This fault is often corrected spontaneously — even more so than other sutural faults — when other cranial faults are corrected and the TMJ is balanced.

Therapy Localization

A previously strong indicator muscle will weaken with positive therapy localization over the squamosal suture; also, a muscle which is weak because of the fault will strengthen. Care must be taken that it is the fault which is actually causing the positive therapy localization, as there are TS line points along this suture which could produce positive therapy localization and be confused with that of the cranial fault. The positive therapy localization could indicate a temporal bulge or a parietal descent cranial fault. Challenge is the best method for determining if the therapy localization is from the cranial fault.

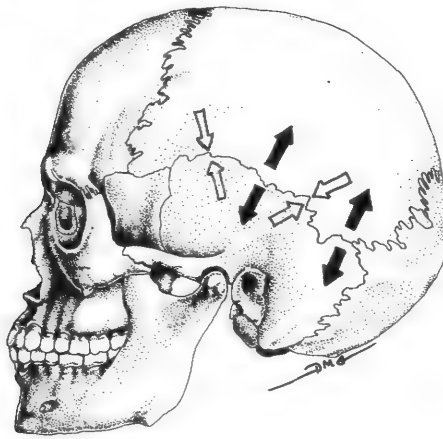
Challenge

Challenge is the method for determining if the cranial fault is present, and also how to correct it. A positive challenge is indicated by a strong indicator muscle weakening. The physician makes a two-handed contact, with one hand on the parietal bone and the other on the temporal. Force is directed to either jam or separate the suture. If the previously strong indicator muscle weakens on approximation the suture is jammed. If the indicator muscle weakens upon separation, the suture is separated and needs further separation for treatment on a rebound basis.

Muscle Correlation

The muscle most significantly involved with this cranial fault is the temporalis. Hypertonicity of the muscle as a result of TMJ dysfunction or other imbalance tends to jam the suture. Other muscles that may be hypertonic are the temporoparietalis and auricularis anterior, superior, and posterior. The

Examination and Treatment of Cranial Faults



6—50. Challenge to separate or jam the suture. Correct in the direction which weakens an indicator muscle on the phase of respiration which abolishes the weakness.

auricularis muscles may be challenged by pulling the ear forward, backward, or inferiorly to determine if the muscles are involved. Challenge of the ear is also sometimes done in auricular therapy as described in Volume III. A positive challenge is signified when a previously strong indicator muscle weakens. If the muscle weakens, a correlation with the cranium can be observed by having the patient take a phase of respiration, either inspiration or expiration. The phase of respiration which abolishes the muscular challenge will be the same as that used in therapy (see below). Also, any dysfunction resulting in tension on the superficial and deep fascia of the epicranium may cause the suture to jam.

Pain Location

Pain along the suture on digital pressure is nearly always present with this cranial fault. There is often a temporal headache which, if due to the cranial fault, will be relieved by holding static pressure to separate the suture.¹⁸ It is hypothesized that the pain is caused by irritation on the nerve endings within the suture. This pattern could also be present in a parietal descent cranial fault.

Conditions Correlating

The temporal headache mentioned above is often associated with a hypertonic temporalis muscle, which may result from temporomandibular joint dysfunction. Whenever this fault is present, the TMJ should be evaluated for possible involvement (see Chapter 12).

Correction

The method of correction is determined by challenge, as described above. Upon finding the challenge — either approximation or separation — that causes a previously strong indicator muscle to weaken, the patient is asked to take a phase of respiration to determine if the indicator muscle strengthens. This will either be on inspiration or expiration. If an approximating challenge weakens the indicator muscle and inspiration abolishes the weakness, correction will be enhanced by having the patient take a deep inspiration as the suture is approximated. Although this is the most common combination, any can be present. For example, the indicator muscle may weaken on separation and strengthen on either inspiration or expiration. In any case, treatment is done on the challenge that weakens a previously strong indicator

muscle while the patient takes the phase of respiration that caused the muscle to regain its strength. It is repeated four or five times, or whatever is required to make the correction.

After the therapeutic attempt, the suture should be challenged and therapy localized to determine if correction was successful. If the correction was for separation, the sagittal suture should be evaluated to make certain it was not jammed during the process of separating the squamosal suture.



6—51. Squamosal cranial fault correction.

LAMBDODIAL SUTURE CRANIAL FAULT

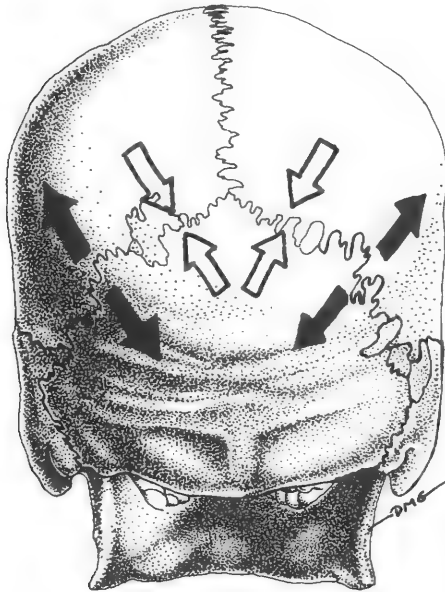
The lambdoidal suture cranial fault is often clinically associated with a closed ileocecal valve syndrome. Like the other sutural cranial faults, it is often corrected secondarily with the correction of one of the more complex faults.

Therapy Localization

There will be positive therapy localization over the lambdoidal suture if the fault is present. Positive diagnosis of the fault is obtained by challenging the suture.

Challenge

When this fault is present, a previously strong indicator muscle will weaken when the suture is separated or approximated to cause further jamming. Challenge indicates the direction of correction, which is done by rebound.



6—52. Challenge to separate or jam the suture. Correct in the direction which weakens an indicator muscle on the phase of respiration which abolishes the weakness.

Muscle Correlation

If the lambdoidal suture is jammed and the fault persists after other, more complex, faults have been corrected, there is probably hypertonicity of the occipitofrontalis; either the occipital or frontal bellies — or both — may be involved. If they are hypertonic, treatment to the proprioceptors is usually indicated. Also, any involvement which causes tension on the superficial or deep fascia of the epicranium must be corrected.

Particular attention should be given to the balance of the upper trapezius and sternocleidomastoid muscles. The rectus capitis posterior (major and minor), semispinalis capitis, and obliquus capitis superior muscles may also be involved.

Pain Location

Typically, there is tenderness along the lambdoidal suture on digital pressure when the fault is present, especially if the suture is jammed.

Conditions Correlating

This cranial fault often correlates with digestive disturbances, especially those clinically associated with the closed ileocecal valve syndrome (see Volume V). It is also often involved when there are whiplash-type injuries.

Correction

The lambdoidal suture is either approximated or separated as indicated by the challenge which caused a previously strong indicator muscle to weaken. This manipulation is done on the phase of respiration which abolished the weakness created by the challenge. The manipulation is done four or five times; then the suture is re-evaluated with challenge and therapy localization to determine if the therapeutic effort was effective. If the involvement is bilateral, each side must be corrected separately.



6—53. Lambdoidal cranial fault correction.

Examination and Treatment of Cranial Faults

ZYGOMATIC SUTURE CRANIAL FAULT

The zygomatic cranial fault is actually three possible cranial faults. It is one or more involvements of the zygomatic bone with the temporal, frontal, or maxillary bones.

Therapy Localization

Therapy localization is done over the temporozygomatic, zygomaticomaxillary, and frontozygomatic sutures. Positive therapy localization, indicated by a previously strong indicator muscle weakening, suggests using challenge for a more positive indication of the sutural cranial fault.

Challenge

Only the zygomatic suture showing positive therapy localization need be challenged. The challenge is the same as that for other sutural cranial faults. Either an approximation or a separation of the suture will cause a previously strong indicator muscle to weaken when the fault is present. The phase of respiration which cancels the positive challenge is then found. The challenge not only differentially diagnoses the fault, it provides information about the corrective approach needed.

Muscle Correlation

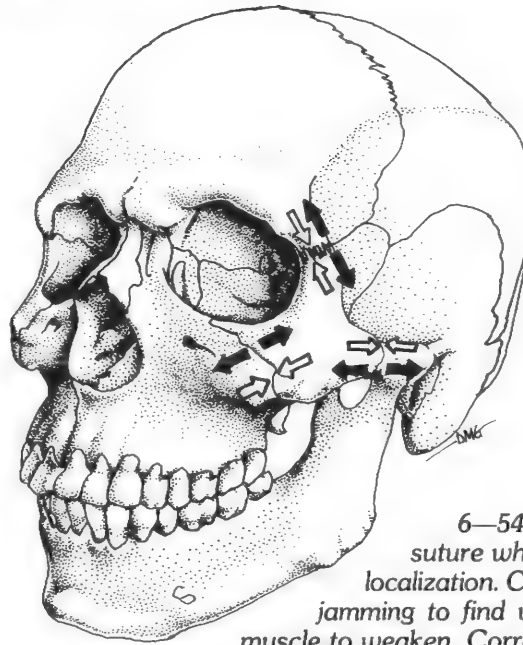
The zygomaticomaxillary suture is most involved with the intrinsic muscles of the skull. The zygomaticus major and minor and the levator labii superioris cross the suture and, if involved, are usually hypertonic.

Pain Location

On digital pressure, pain may be located over any one of the sutures. A spontaneous facial pain may be associated with the involvement.

Conditions Correlating

On a clinical basis, the zygomatic suture cranial faults are often associated with an open ileocecal valve syndrome. Although this syndrome is routinely associated with these particular faults, any cranial fault may be involved.



6—54. Challenge the zygomatic suture which shows positive therapy localization. Challenge for separation and jamming to find which causes an indicator muscle to weaken. Correct in the direction of positive challenge on the phase of respiration which abolished the weakness.

Correction

One or more of the zygomatic sutures may be involved; each is evaluated and treated separately. Treatment consists of a two-handed contact on either side of the suture to either approximate or separate the suture as indicated by challenge. Therapeutic pressure is applied on the phase of respiration which caused the indicator muscle to regain its strength. The effort is repeated four or five times; then the suture is re-evaluated with therapy localization and challenge to determine if correction was effective.



6—55. Correction of temporozygomatic suture.



6—56. Correction of frontozygomatic suture.



6—57. Correction of zygomaticomaxillary suture.

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Chapter 7

Examination and Treatment of Pelvic Dysfunction

Introduction

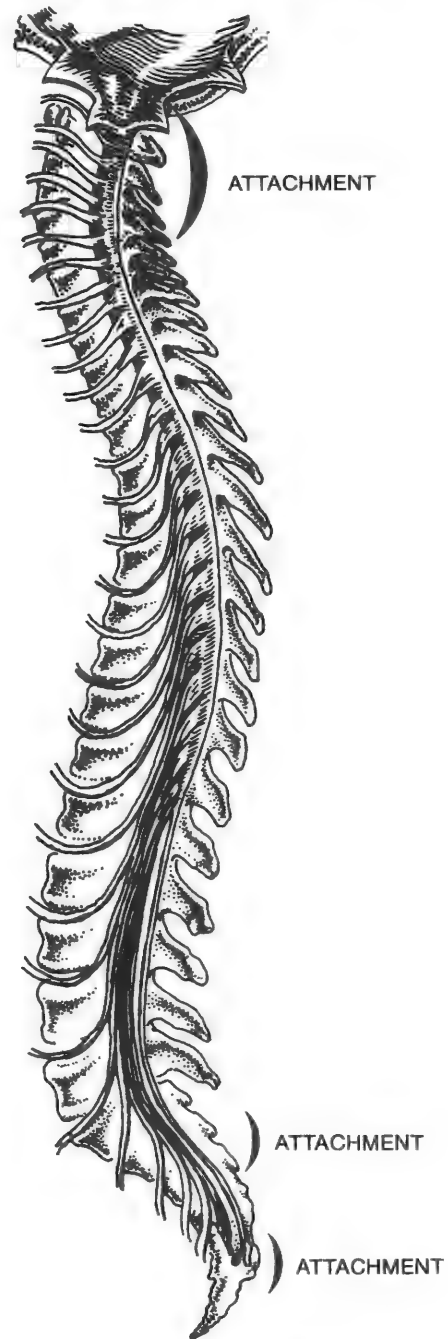
Pelvic and cranial motion are closely integrated. Sacral motion — like that of the cranium — is independent of thoracic respiration but can be influenced by it. Continuity between the cranium and sacrum is by way of the dura mater, which is firmly attached at the foramen magnum and to the 2nd and 3rd cervical vertebrae. Another view is taken by DeJarnette and Denton,³ who indicate that the dura "... attaches to the outer two-thirds of the ring of the atlas." It does not have firm attachment again until the 2nd sacral segment. From here the dura mater invests the filum terminale of the spinal cord and descends to the back of the coccyx, blending with the periosteum.²¹

The innominates also have a motion which appears independent but is greatly influenced by the muscles of thoracic and diaphragmatic respiration. These muscles are very important in pelvic function, which in turn relates with cranial function. Postural muscles also must function correctly for normal pelvic motion.

Whenever there is a cranial fault, it is mandatory that the pelvis be examined. There is often aberrant motion, or lack of motion, in the pelvis which is in harmony with the cranial faults. If the cranium is corrected and the pelvis is not, cranial faults will probably return; this usually happens as soon as the patient walks.

The pelvis is generally examined, and corrected if necessary, immediately after the cranium is corrected. It does not seem to matter which is corrected first, or if both are corrected simultaneously.

In Volume I of this series, the category I pelvic fault is discussed regarding its examination and correction. In this volume there is some repetition of that material, but here the mechanics and correction of the respiratory nature of the category I pelvic fault are considerably expanded. Additional pelvic function — including the sacral and innominate primary respiratory function — and relation to the muscles are discussed. In addition to the examination and correction of the category I pelvic fault, examination and correction procedures for sacral faults, sacral wobble, atlanto-occipital counter torque, and coccygeal dysfunction are also presented.



7—1. Dural attachments.

Examination and Treatment of Pelvic Dysfunction

Pelvic Anatomy

SACRUM

The sacrum is composed of five sacral vertebrae or segments which ossify at approximately twenty to twenty-five years of age to form one structure. The apex of the bone is inferior and articulates with the coccyx. The base of the structure is superior and articulates with the 5th lumbar vertebra, forming the sacrovertebral angle. In addition to the base and apex, the sacrum has four surfaces — the dorsal, pelvic, and two lateral surfaces. The center is hollow, making up the sacral canal.

The base is formed by the first sacral segment and presents the sacral promontory, which is the anterior projecting edge. Posterior are the superior articular processes, which articulate with the inferior articular processes of the 5th lumbar vertebra. The upper lateral part of the sacrum is a broad, flared mass called the "ala."

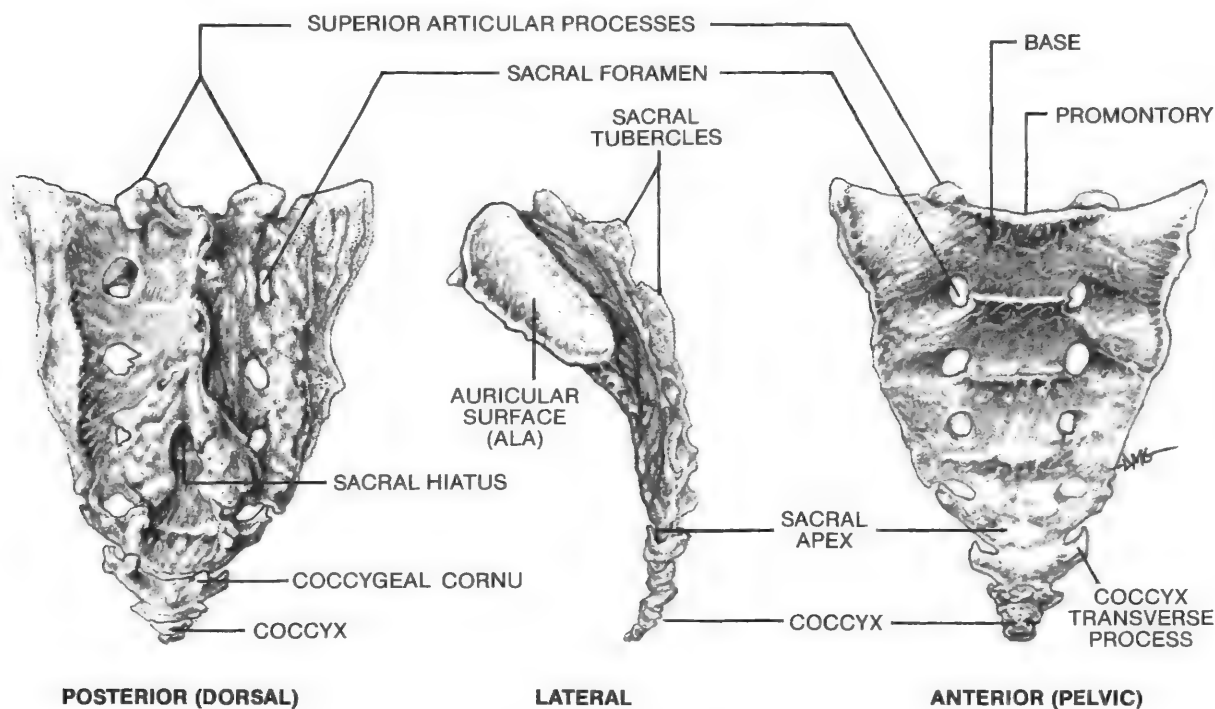
The pelvic surface is concave and faces anteriorly and inferiorly. The four bilateral sacral foramina communicate with the sacral canal and transmit the ventral rami of the upper four sacrospinal nerves.

The dorsal surface is convex and faces posteriorly and superiorly. In the center are three or four sacral tubercles which are equivalent to the spinous processes of the vertebrae. On each side of the sacral tubercles are four foramina, communicating

with the sacral canal for transmission of the dorsal rami of the sacrospinal nerves. The superior sacral segment has two processes which articulate with the 5th lumbar. At the inferior, or apex, of the sacrum are the sacral cornua for articulation with the coccygeal cornua.

The lateral surface of the sacrum presents an ear-shaped surface for articulation with the ilium of the innominate bone, making the sacroiliac articulation.

The sacral canal traverses the entire length of the sacrum, with openings anterior and posterior for the sacral foramina. Inferiorly the opening is called the sacral hiatus. Contained within the upper portion of the sacral canal are the arachnoid mater and dura mater; these are extensions of the dural and arachnoid meninges, with a firm attachment at the 2nd sacral segment. Within the dura and arachnoid is the filum terminale internum, continuing from the apex of the conus medullaris. This fine filament is a continuation of the pia mater of the spinal cord. As the filum terminale emerges from the arachnoid mater and dura mater, it is called the filum terminale externum; it continues to descend to attach to the dorsum of the 1st coccygeal segment.



7—2. Sacrum and coccyx.

COCCYX

The coccyx is triangular and made up of three to five rudimentary vertebrae which may be fused together. The superior and largest segment has a base which articulates with the apex of the sacrum.

Projecting from the base are the coccygeal cornua, which articulate with the sacral cornua. A rudimentary transverse process projects laterally from each side of the 1st segment.

INNOMINATE BONE

The innominate has three major parts — the ilium, ischium, and pubis. In the infant the three parts are connected by cartilage, ossifying into one structure to make up the innominate bone. The innominate has a cup-like cavity for articulation with the head of the femur. The three sections of the innominate join in this area. The ischium and pubis also join below the acetabulum to form the obturator foramen.

The *ilium* is described as having two parts — the body and the ala. The section containing a portion of the acetabulum is the body, and above it is the ala.

The body forms approximately two-fifths of the acetabulum. It is where the ilium joins the ischium and pubis, giving little indication of the place of union in the adult innominate bone.

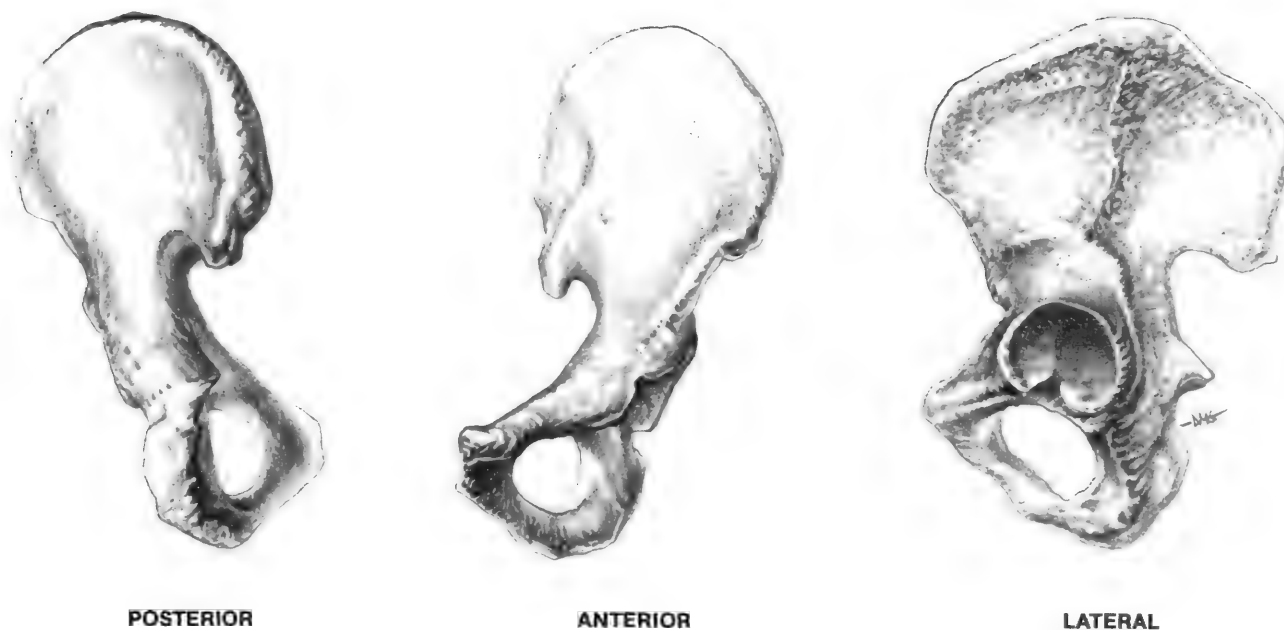
The ala has two surfaces, a crest, and two borders. The external surface is primarily the attachment point for the gluteus medius and minimus. The upper portion of the external surface presents the crest of the ilium. The anterior and posterior limits of the crest are referred to as the anterior and posterior superior iliac spines. Along the crest of the ilium are the attachment points for the oblique and transverse abdominal muscles. As will be seen later, these attachment points provide considerable leverage for these muscles to influence pelvic motion. The ventral border of the ala continues down from the anterior superior iliac spine. At this point the tensor fascia lata muscle originates. Immediately below is a notch from which the sartorius muscle originates. Below the notch is the anterior inferior iliac spine, giving origin to the straight head of the rectus femoris muscle. Medial to the anterior inferior iliac spine is a broad, shallow groove over which the iliacus and psoas major pass. On the medial side of the groove is the iliopectineal eminence, which marks the point of union of the ilium and pubic sections of the innominate bone.

The dorsal border of the ala is bounded superiorly by the posterior superior iliac spine, which

provides an attachment point for the multifidus muscle and the dorsal sacroiliac ligaments. The lower aspect of the dorsal border is bounded by the posterior inferior iliac spine, inferior to which is the greater sciatic notch. The internal or pelvic surface of the ala presents a large, concave surface called the iliac fossa which gives origin to the iliacus muscle. Medial and posterior to the iliac fossa is the auricular surface, which articulates with the sacrum to form the sacroiliac articulation.

The *ischium* is divided into a body and a ramus. The body has upper and lower sections and demonstrates femoral, dorsal, and pelvic surfaces. The upper section of the body contributes to the formation of the acetabulum, while the lower section gives off the ramus. The ramus joins with the pubis to complete the obturator foramen. The combined rami are sometimes called the ischial pubic ramus. The ischial tuberosity is a large section which comprises the inferior aspect of the ischial body. The hamstrings originate from this tuberosity. A pointed triangular eminence called the ischial spine extends from the dorsal border of the body; the gemellus superior, coccygeus, and fibers of the levator ani muscles, and the pelvic fascia and sacrospinous ligament arise from it. Superior to the spine is the greater sciatic notch; inferior is the lesser sciatic notch. The ramus of the ischium presents anterior and posterior surfaces. The anterior surface provides attachment for some of the medial femoral muscles.

The *pubis* is divided into a body and a superior and inferior ramus. The body forms one-fifth of the acetabulum. The obturator internus arises on its internal surface. The superior ramus extends from the body medially to articulate with the opposite superior ramus. From the crest of the ramus arises the rectus abdominis muscle, and from the anterior surface arises the pyramidalis muscle. The inferior ramus passes posteriorly, inferiorly, and laterally to unite with the ramus of the ischium on the medial side of the obturator foramen.



7—3. *Innominate bone.*

SACROILIAC ARTICULATION

The surfaces of the sacrum and innominate bone which make up the sacroiliac articulation are shaped somewhat like an ear, thus the name auricular surfaces. There are two main elevations on the sacral surface which are located cephalad and caudally. The iliac surfaces are reciprocally shaped, but they are not exact mirror images. With age the elevations and depressions of the articular surface increase.^{16, 19}

Weisl¹⁸ describes the ligament suspension of the sacroiliac articulation. The sacrum and ilium are held in apposition by a capsular ligament and a number of accessory ligaments. There are two distinct groups of fasciculi which support weight in the standing position. Illi¹¹ discovered a ligament within the sacroiliac articulation. It lies primarily within the synovial membrane, originating by three or four digitations at a point about 12 mm above the superior border of the articulating surface of the ilium. Fasciculi converge to enter the joint capsule.

There appear to be two types of movement at the sacroiliac articulation. There is a gross movement associated with weight bearing which is very active in movements from the supine to sitting position, walking, bending, and twisting. Motion of this nature at the sacroiliac has been denied vigorously by some authorities in the past; most now recognize it. Gray's Anatomy (British edition)²¹ acknowledges a small amount of anteroposterior rotatory movement about

a transverse axis, which is usually about 5-10 cm vertically below the promontory of the sacrum. They go on to comment on Weisl's research¹⁷ that the "... greatest change in position of the sacrum relative to the iliac bones occurs when rising from the recumbent to the standing position. The sacral promontory moves forward as much as 5-6 mm as the body weight is taken upon the sacrum." Weisl also observed no difference in sacroiliac motion between males and females, with the exception of the puerperal state. Women who were four to ten days postpartum showed a greater range of motion in the articulation.²⁰ In a roentgenographic study of 111 young adults, Weisl¹⁵ found that 90% of the subjects demonstrated movement in the conjugate diameter of the pelvis. In yet another study,¹⁹ he noted that aging of the subject was associated with roughening and fissuring of the articular cartilage. Near the dorsal border an elevation is formed which is sometimes of considerable size. These changes diminish the mobility of the joint, possibly culminating in its ankylosis.

Gray's Anatomy (American edition)⁹ is less specific about pelvic motion, stating that the cartilaginous plates of the sacrum and ilium are "... separated by a space containing a synovial fluid, hence the joint presents the characteristic of a gliding joint." The study of Frigerio et al.,⁴ discussed in Volume I,

appears to definitively document sacroiliac motion.

There appears to be a second type of motion at the sacroiliac articulation related to the cranial-sacral primary respiratory movement. Most of the auricular surface angles so that the right and left surfaces diverge anteriorly and converge posteriorly.¹² The single area of auricular surface not so angled is at the area considered the articulating surface for the

primary respiratory mechanism. This area angles so that the right and left surfaces converge anteriorly and diverge posteriorly, making this single area of the surface correlate with the divergent articulations represented throughout the cranium.¹² The general axis of primary respiratory rotation of the sacrum, then, appears to be at the 2nd sacral segment level.

Primary Respiratory Motion of the Pelvis

Each pelvic bone has a specific movement correlated with sphenobasilar flexion and extension. The sacrum and coccyx appear to have primary respiratory movement relating with dural tension from cranial movement. The innominate bones appear to move more significantly with thoracic respiration, depending upon the abdominal muscles for their movement.

There is an interdependent action between the sacrum, coccyx, and the innominate bones. It appears that the primary respiratory movement of the sacrum and coccyx influences the innominate bones; on the other hand, the more dramatic movement of the innominates from thoracic respiration influences the sacrum and coccyx. It may be from this innominate activity acting on the sacrum that thoracic respiration primarily influences the cranium through the connection of the dura mater.

Michael and Retzlaff¹³ demonstrated parietal bone movement in an anesthetized monkey when its spine was flexed. This activity may be correlated either to change of cerebrospinal fluid pressure or to tension on the dural membranes. Both activities have been hypothesized to influence the cranial-sacral

primary respiratory mechanism. It is possible that induced pelvic motion from thoracic respiration may influence the cranium in a manner similar to the experimental spine flexion.

In a postural roentgenographic evaluation of seventeen individuals with a low occiput on one side, Greenman¹⁰ found fifteen with a low sacrum on the same side. This gave correlation of 89% who fit the Lovett Brother principle. The two who did not were not evaluated further. There may have been congenital variances in these two subjects. Another factor may have been neurologic disorganization causing imbalance of the muscles which support the head-on-body and pelvic balance.

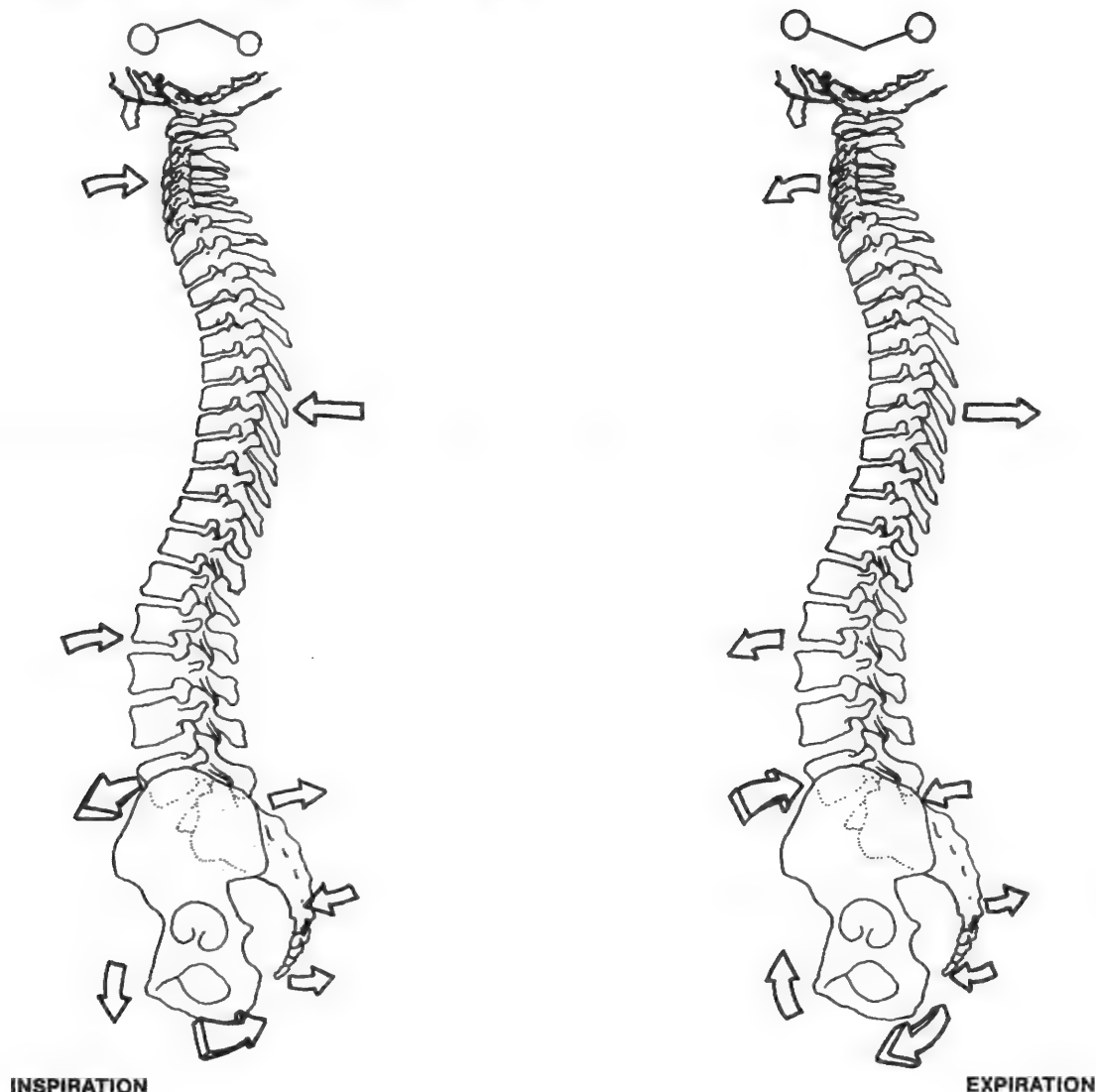
The innominates move considerably with thoracic inspiration, the lumbar spine flattens, and the sacral apex moves forward and its base posteriorly. These motions correlate with an elongation of the dura to move with sphenobasilar flexion, which is present on inspiration. The opposite movements are present on exhalation, shortening the dura to pull the cranium into sphenobasilar extension. Consideration of individual bone movement, and then placing all activities together, helps clarify this activity.

SACRAL RESPIRATORY MOVEMENT^{5,7}

Sacral respiratory movement is in the sagittal plane. The axis of rotation is in the general area of the 2nd sacral segment about the transverse axis. The primary respiratory motion of the sacrum on sphenobasilar flexion is for the apex of the sacrum to move anteriorly and its base posteriorly and superiorly. Motion is due to the elevation of the sphenobasilar junction and consequent elevation of the dura, lifting and bringing the base posteriorly while throwing the

sacral apex anteriorly. Thoracic inspiration augments this activity. As the diaphragm contracts and applies pressure to the abdominal contents, the lumbar spine moves posteriorly and reduces the lumbar lordosis. This flattening of the curve carries the base of the sacrum posteriorly, moving its apex anteriorly. The sacrum and lumbar move opposite on sphenobasilar extension or expiration.

The sacrum and occipital bone are considered



7—4. Motion of sphenobasilar junction, spine, sacrum, coccyx, and innominate bone on inspiration and expiration.

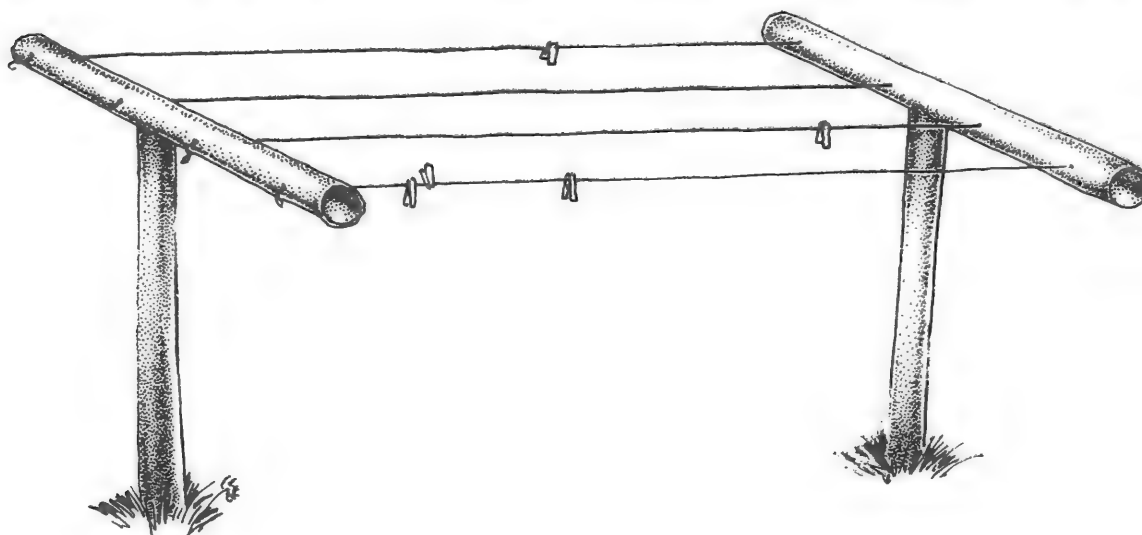
Lovett Brothers. The term “Lovett Brother” refers to structures remote from each other which function and also misalign in a similar manner, with one misalignment accommodating the other. (For a more complete discussion of “Lovett Brother,” refer to Volume I.) If the sacrum misaligns inferiorly on one side, the occipital bone will likely be inferior on the same side. Likewise, if there is an inspiration assist cranial fault on one side of the cranium, there will probably be an inspiration assist in the pelvis on the same side, if the pelvis is involved. In cranial osteopathy, dural communication between the cranium and sacrum is called the “core-link.” Magoun¹² uses the illustration of the dura as cables — rather than a tube — connecting the occipital bone and sacrum. With balance there will be equal tension on both sides. If the sacrum moves, it produces tension

on one cable, thus pulling the occipital bone into a similar position. This model, for illustrative purposes, is further presented as clotheslines connecting two poles with cross-arms. If one pole rotates, it will rotate the other pole in a similar manner, since together with the clotheslines they make up a functional unit.

Sacral involvements are listed as inspiration or expiration assists. An inspiration assist lesion means that the sacral apex is not moving forward enough on sphenobasilar flexion. The involvement can be either unilateral or bilateral. Because the cranial-sacral primary respiratory mechanism is a functional unit, it should operate in a predictable manner. If there is an inspiration assist cranial fault on the right, there should also be an inspiration assist sacral fault on the right, if the sacrum is involved. There should not be

an inspiration assist cranial fault on one side and a sacral inspiration assist on the other. If examination reveals this lack of coordination, some factor is causing neurologic disorganization; the body is operating in an improper manner which may be caused by a functional disturbance or body interfer-

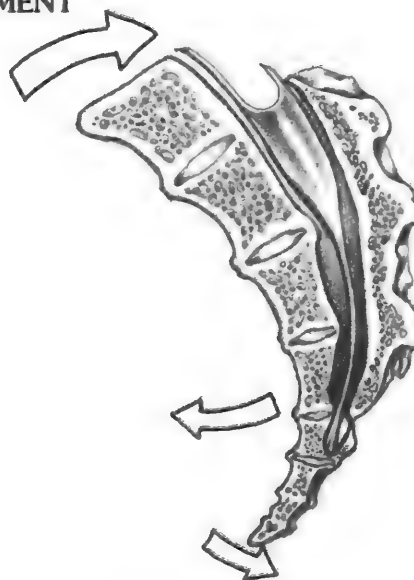
ence, making the examination information confused. The patient should be evaluated for switching as presented in Volume I. After correction of the switching, the pattern of the cranium and sacrum should correlate. (A more advanced approach to switching is presented in Section III of this volume.)



7—5. If one pole is rotated, the other will rotate in a similar manner. This is similar to the "core-link" of the cranial sacral primary respiratory system.

COCCYX MOVEMENT

On sphenobasilar flexion, the apex of the coccyx appears to move posteriorly and its base anteriorly. This is exactly opposite the sacral movement. The precise mechanics of coccygeal movement are unknown. It may move in this manner because the filum terminale is attached posteriorly to the general axis of rotation of the coccyx. The hypothesis of movement is derived from restricting the motion of the coccyx on different phases of respiration, and then testing a previously strong indicator muscle to determine weakening. If the coccygeal movement follows the pattern of other bones in the cranial-sacral mechanism, its movement is as described. The coccyx functions in a Lovett Brother arrangement with the sphenoid. If the sphenoid moves superiorly on the right, the coccyx will most likely move superiorly and laterally on the right. This correlative motion is readily observed by challenging the two structures. This again should correlate in examination; failure to do so probably indicates switching. The hypothesis of coccygeal movement seems to be enhanced by observation of the Lovett Brother arrangement between the coccyx and the sphenoid. First, consider that the occipital bone and the sacrum rotate in the same direction on inhalation and



7—6. Inspiration of sacrum and coccyx.

on exhalation, as do the sphenoid and coccyx. It is interesting to observe that the rotation of the occipital and sphenoid bones is opposite each other, and so is that of the sacrum and coccyx; this seems to enhance the Lovett Brother concept.

INNOMINATE MOVEMENT

As mentioned, the innominate bones move with thoracic respiration and probably influence the sacrum. Movement of the innominate bones depends upon the abdominal muscles. As the diaphragm contracts upon inhalation the abdominal muscles are inhibited, giving less support to the innominate. The symphysis pubis moves inferiorly and posteriorly. The anterior superior iliac crest moves laterally and inferiorly, while the ischium moves posteriorly and medially.

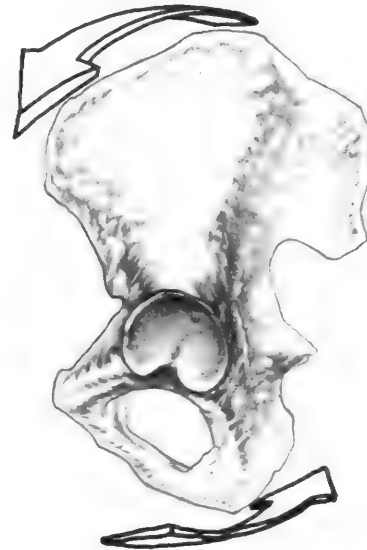
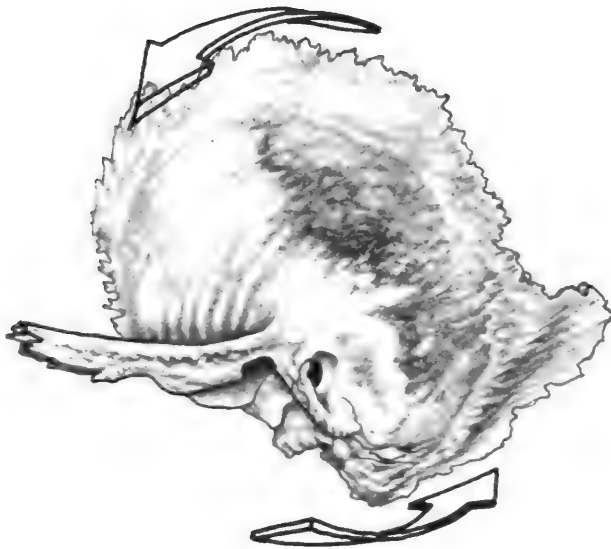
During expiration the abdominal muscles contract as the diaphragm relaxes, causing motion opposite that described for inspiration.

The innominate and temporal bones have analogous parts in reference to motion. The crest of the ilium is analogous to the squama of the temporal bone, the mastoid process to the ischial tuberosity, and the zygomatic process to the symphysis pubis.

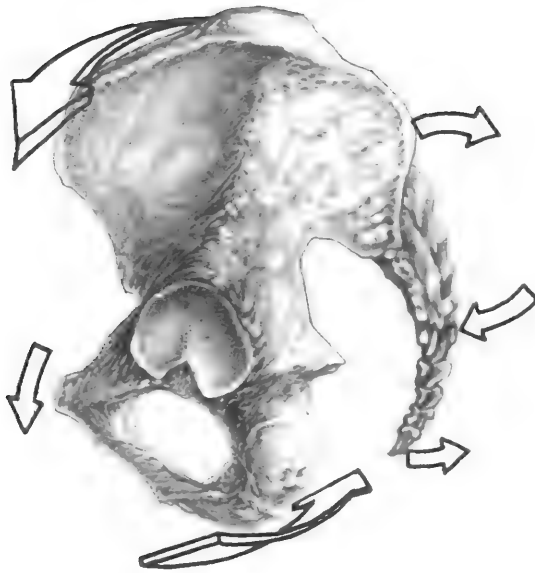
Although the innominate bone moves in the same way as the temporal, it does not have the same positive correlation that the sacrum does with primary respiratory movement. This is because the activity of the innominate bones is primarily from the abdominal musculature. If one or more of the abdominal muscles is imbalanced, there will be an influence on innominate motion which may override

the cranial-sacral primary respiratory activity. The abdominal muscles have a considerable amount of leverage on the sacroiliac articulation because of their insertion on the ilium and pubis. It can easily be seen how important abdominal muscle balance is in obtaining normal pelvic function; if balance is not present, the cranial-sacral primary respiratory mechanism may be adversely affected.

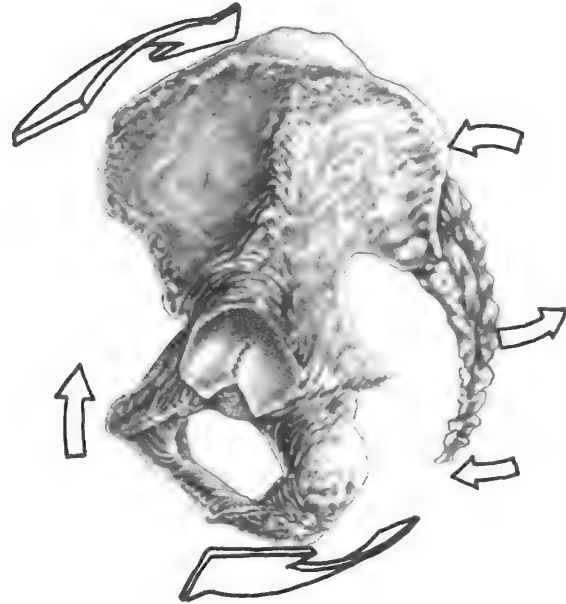
In addition to the influence of the abdominal musculature on the innominate bones, the structure is related to the weight-bearing mechanism. Imbalance of postural muscles — such as the psoas, sartorius, gracilis, hamstrings, and others — may cause innominate dysfunction to create a sacroiliac subluxation, or dysfunction of the innominate movement relating with primary respiration. The innominate may thus create an imbalance in sacral primary movement, which in turn influences the cranium. For this reason, sacral primary respiratory involvement when a patient is prone or supine may not be observed, but it will be seen in a weight-bearing position. It has already been pointed out how postural muscles — the sternocleidomastoid, upper trapezius, and others — can adversely influence the cranial primary respiratory mechanism.



7—7. Movement of temporal and innominate bones on inspiration (sphenobasilar flexion).



INSPIRATION



EXPIRATION

7—8. Movement of innominate, sacrum, and coccygeal bones on inspiration and expiration.

Pelvic Primary Respiratory Examination and Treatment

The types of pelvic involvements which deal directly with the primary respiratory mechanism are inspiration and expiration sacral assist, coccygeal

involvement, category I, and a specialized sacral involvement called sacral wobble, which is often correlated with an atlanto-occipital counter torque.

SACRAL INSPIRATION ASSIST

Mechanics of the Fault

Abnormal position of the sacrum occurs when the apex is posterior and the base is anterior. Inspiration brings the sacrum into an improved position and causes a muscle weak as a result of the fault to strengthen. Expiration puts the sacrum further out of normal position and causes a previously strong indicator muscle to weaken. The hamstrings are usually evaluated for a sacral fault; they may either be weak as a result of the fault or strong in the clear. If the hamstrings are weak in the clear and related to sacral inspiration assist, they will

strengthen when a deep inspiration is taken and held. Obviously other factors — such as neurolymphatic, neurovascular, subluxations, meridian imbalance, etc. — can cause the weak muscle. These are evaluated with therapy localization and challenge as described in Volumes I and III. Correction of these factors may make the hamstrings strong in the clear; they can then be used to evaluate for sacral involvement as if they were strong in the clear at the beginning. If the hamstrings are strong in the clear, the patient exhales and holds that phase of respiration while the hamstrings are tested. They will weaken if there is a

Examination and Treatment of Pelvic Dysfunction

sacral inspiration assist fault.

Although the primary motion of the sacrum is flexion and extension in the sagittal plane, the sacrum becomes misaligned with the same three-dimensional characteristic present in the cranial bones. The rotational aspects of the sacrum are probably influenced by innominate balance and other factors in the weight-bearing position.

Mechanics of Correction

Correction of a sacral primary respiratory fault is best done with a direct force. A rebound approach may occasionally unlock the mechanism, but it is not as consistent a correction as the direct method. As the patient takes a deep inspiration, the physician applies pressure to the apex of the sacrum in the direction indicated by challenge. Some physicians apply pressure to the base of the sacrum as the patient exhales. This additional maneuver is not usually necessary to obtain effective correction.

Therapy Localization

The fault will generally therapy localize over the sacroiliacs or over the sacrum. Although therapy localization is not pathognomonic of the condition, it indicates that the fault may be present. Challenge should then be used to determine if, in fact, there is a sacral fault and what type it is. After correction, therapy localization is a good method for determining the effort's effectiveness.

Therapy localization can be used in conjunction with a respiratory challenge to find hidden sacral faults. The patient therapy localizes in the usual manner to the sacrum, and then a previously strong indicator muscle (usually the hamstrings) is tested for weakening on complete expiration or inspiration. If the indicator muscle weakens on expiration, the involvement is an inspiration assist fault; if it weakens on inspiration, it is an expiration assist fault.

Challenge

The sacrum is challenged at its apex in an anterior direction. The pressure is released, and a previously strong indicator muscle is tested for weakening, or a weak muscle associated with the fault is tested for strengthening. Application of force can be either on the right or left side of the apex — correlating with a right or left fault — or in the center. Various vectors of force should be used to find the maximum weakening of the previously strong indicator muscle. A vector may be applied with a medial, lateral, superior or inferior component added to the anterior force. It may be necessary to apply a torque with the anterior force to obtain maximum weakening of a strong indicator muscle. The vector which

makes the maximum change is the optimum one for correction.

Muscle Correlation

The piriformis muscle is often directly related with the sacral primary respiratory fault. There will usually be weakness of the piriformis on the side of the sacral fault. The piriformis will often fail to weaken in the prone or supine position, but it will weaken in a weight-bearing position. It can be tested with the patient standing or in the hands-and-knees position. When testing the piriformis in a standing position, it is best to have the patient lean against an upright hi-lo table. A more even weight distribution into the pelvis can be obtained with the patient in the hands-and-knees position. The knee and hip are flexed 90°, and the piriformis is tested for its rotational capability. For optimum results, it is best to strengthen the piriformis prior to sacral correction.

The psoas major or iliacus muscles may be out of balance with each other or with the rest of the body. They relate to the sacrum because of crossing the sacroiliac articulation.

Pain Location

There may or may not be sacroiliac pain associated with the sacral fault. The pain may be spontaneous or elicited only on digital pressure.

Conditions Correlating

If the cranium is involved with an inspiration sacral fault, it will often have an inspiration-type involvement on the same side. If there is a left sacral inspiration assist, there will usually be a left inspiration assist, sphenobasilar inspiration assist, or temporal bulge. Failure of the sacral-pelvic faults to correlate with those of the cranium indicates there is probably some form of neurologic confusion present, but it does not always mean neurologic disorganization will be found. There can be congenital differences or structural change from trauma, surgery, etc., which may cause the lack of correlation. If the findings fail to correlate, the patient should be evaluated for switching and other factors to determine the reason.

If there is a temporal bulge, the maximum strengthening of a weak muscle associated with a sacral fault will often be on one-half breath inspiration held.

Correction

Four or five pounds of pressure should be applied to the apex of the sacrum in the direction of optimum challenge. The patient should inhale from complete expiration to complete inspiration. As with correction of inspiration assist cranial faults, the pressure



7—9. *Inspiration assist correction. First challenge for optimum vector and then correct with patient's inspiration until freedom of motion is developed.*

should be continued throughout the full phase of respiration, with the patient inspiring slowly. Generally four or five repetitions will eliminate the involvement. When correction is obtained, the physician should feel an improved movement of the sacrum.

After the therapeutic attempt, the sacrum should be re-evaluated with therapy localization and challenge; any muscles weak as a result of the fault should be strong. There should be no weakening of an indicator muscle on full expiration.

SACRAL EXPIRATION ASSIST

Mechanics of the Fault

The expiration assist sacral fault occurs when the sacral apex is too far anterior and the base posterior. A muscle which weakens as a result of the fault will become strong while the patient holds his breath in complete exhalation. A previously strong indicator muscle will become weak on full inspiration.

The fault may be unilateral, bilateral, or combined with a sacral inspiration assist fault on the other side. The involvement may be only in the sagittal plane, but more often it is three-dimensional.

Mechanics of Correction

As with the inspiration assist sacral fault, this fault is corrected directly. The examiner contacts the anterior aspect of the sacral apex immediately above the coccyx, usually with a thumb, to move the apex

posteriorly as the patient exhales. The physician may want to use a two-handed approach to contact the base of the sacrum, moving it anteriorly during the same exhalation.

Therapy Localization

Therapy localization is over the sacroiliac articulations or over the sacrum. It should correlate with challenge and muscle testing on various phases of respiration to give positive indication of the fault. The same type of combined therapy localization and respiratory challenge described for the inspiration assist sacral fault can be used with the expiration assist. Therapy localization does not distinguish the specific type of sacral involvement present; it simply indicates that something is involved. It may be a subluxation of the sacrum or innominate, a category

Examination and Treatment of Pelvic Dysfunction

I, or an inspiration assist. Challenge and breathing correlation must be done to differentiate this fault.

Challenge

With the patient prone, the physician's thumb is used to contact the anterior aspect of the sacral apex to challenge in a posterior direction. A two-handed challenge is sometimes used, with the other contact on the posterior sacral base. Various vectors are used, whether the challenge is one- or two-handed, to find the maximum weakening of a previously strong indicator muscle. This is the best vector for correction.

Sometimes it is necessary to challenge the sacrum in a weight-bearing position. This is accomplished with the patient leaning the ventral surface against a hi-lo table. The patient can also be in a hands-and-knees position. Challenge of the sacrum is done by the examiner exactly as if the patient were lying prone.

Muscle Correlation

Imbalance of the piriformis muscle is also correlated with the expiration assist sacral fault. In fact, the piriformis may be involved with any type of pelvic involvement relating to sacral position.

The psoas major muscles should be evaluated since they also cross the sacroiliac articulations. The balance of the sacroiliac articulations may be influenced by either the psoas, iliacus, or piriformis, especially if there is hypertonicity on one side and hypotonicity on the other.

Pain Location

There may be pain over the sacroiliac articulations, either on digital pressure or spontaneously. In some instances, the patient will complain of discomfort in the posterior aspect of the pelvis when taking either a deep inspiration or expiration.



7—10. The arrows indicate the point of contact to challenge or correct the sacral apex in a posterior direction for expiration assist fault. Usually challenge indicates optimum correction is from contact on only one side. Correct with expiration four or five times, or until motion is felt.

Conditions Correlating

If a cranial fault is present, it usually is an expiration assist on the side of sacral expiration assist. This would include an expiration assist, sphenobasilar expiration assist, or parietal descent cranial fault. If the cranial involvement is primarily a parietal descent, maximum strengthening of the weak associated muscle(s) with the sacral fault will be on one-half breath expiration held.

Correction

When correcting the expiration assist sacral fault, it is necessary to contact the sacral apex immediately above the coccyx as far anteriorly as possible. This is most easily accomplished by elevating the pelvic piece of the adjustment table. This also probably aids the correction by influencing the weight-bearing proprioceptors of the skin, discussed in Volume I. The physician contacts the sacral apex and lifts it posteriorly in the direction that caused the maximum weakening of a previously strong indicator muscle on challenge. If the challenge was done with two hands, the base of the sacrum is contacted with the other hand and pressed in the direction of maximum positive challenge with four or five pounds of pressure. Force is applied through the full phase from inspiration to expiration, with the physician holding a steady pressure the entire time. If the patient attempts to breathe too rapidly, the procedure should be slowed down. Generally four or five repetitions are adequate. The physician will



7—11. Two-handed correction of expiration assist. The physician's thumb lifts the apex of the sacrum while the superior hand moves the sacral base anteriorly.



7—12. Expiration assist. Challenge first for optimum vector of correction.

usually be able to feel the sacrum regain normal motion when the therapeutic attempt is successful.

After the corrective attempt, re-evaluate with therapy localization and challenge, and by testing the indicator or weak muscle with respiration.

Bilateral Involvement

There may be an inspiration assist and an expiration assist sacral fault on opposite sides. This is not as common as finding opposites in the cranium. If the opposite conditions are found, they can be treated simultaneously. First, challenge each side to determine the correct vector; then press anteriorly on the sacral apex with inspiration and draw it posteriorly with expiration. The same evaluation after the corrective attempt should be done as with individual faults.



7—13. Combination inspiration and expiration assist administered alternately as patient inhales and exhales.

COCCYX

A primary coccygeal fault is generally due to trauma, such as a sit-down type of fall, or improper sitting positions which continue to strain the structure. The coccyx may be involved secondarily to a sphenoidal fault, in which case its position — as indicated by challenge — will usually duplicate that of the sphenoid.

The coccyx is nearly always involved in the primary respiratory mechanism when coccygodynia is present. The primary respiratory involvement may be in addition to actual subluxation of the structure. The articulations of the coccyx may be sprained or strained, or have degenerative arthritis. The possibility of local tumors should be considered, as well as lumbosacral lesions or some other cause.¹⁴

Therapy Localization

When the coccyx is involved with either a subluxation or the primary respiratory mechanism, there will be positive therapy localization at the sacrococcygeal articulation. It is difficult to find a muscle weak as a result of coccygeal disturbance, so it is generally best to evaluate a strong indicator muscle for weakening upon therapy localization. If there is weakness of some postural muscle as a result of a coccygeal fault, it will strengthen on therapy localization to the sacrococcygeal articulation.

Challenge

The coccyx is challenged primarily for a combination of laterality and movement in an anterior or posterior direction. It is sometimes necessary to challenge the coccyx via the rectum. A previously strong indicator muscle will weaken on manual muscle testing in the presence of a positive challenge. Since the coccyx responds to a rebound challenge, it is corrected in the direction of positive challenge.

Muscle Correlation

The levator ani and coccygeus muscles are often involved with coccygeal faults. The muscles are usually imbalanced from one side to the other. The involvement of the levator ani is often weakness, while the coccygeus appears to be hypertonic. The applied kinesiology approach to the levator ani muscle is discussed in Volume V.

Pain Location

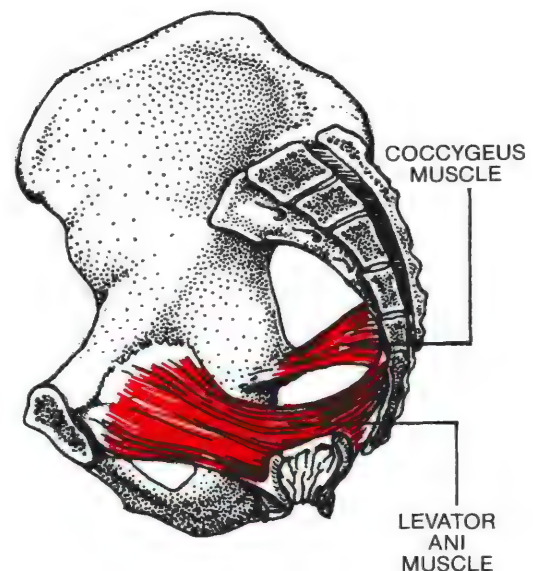
Coccygodynia may be localized or general in the coccygeal-sacral area. It is more often localized and may be aggravated by bowel movement, sitting, lying supine, or other activities causing movement or strain to the articulation.

Conditions Correlating

The sphenoid is the Lovett Brother of the coccyx; it is often in lesion in the same position as the coccygeal lesion. A sphenoidal fault may relate to any of the following faults: inspiration/expiration assist, sphenobasilar inspiration/expiration assist, frontal rotation, or temporal bulge and parietal descent. If any of these are present, correction of the cranium prior to an attempt to correct the coccyx aids the coccygeal correction. Immediately after correcting the cranium, the coccyx should be corrected with minimal patient movement; coccygeal involvement may re-create the cranial fault.

Correction

The coccyx can be adjusted with a standard adjustive thrust. The amount of force should be light because of the structure's fragile nature. An ideal method for correcting most coccygeal subluxations is with a respiratory assist adjustment. The physician presses on the coccyx in the direction which caused maximum weakening of a previously strong indicator muscle, on the phase of respiration which abolished the weakness produced by challenge. The pressure is repeated with four or five appropriate respirations, and the coccyx is re-evaluated with therapy localization, respiratory assist, and challenge to assure that correction was obtained.



7—14.

CATEGORY I PELVIC FAULT

The category I pelvic fault is discussed in Volume I. The approach here will give a brief review of that material, but it will correlate more with the primary respiratory nature of the category I.

The category I pelvic fault relates to a pelvic twist, but it is not an osseous lesion or subluxation of the sacroiliacs (the category II pelvic subluxation). This pelvic involvement requires a different approach from that of the sacroiliac or symphysis pubis subluxation. Both the category I and category II pelvic faults can adversely influence the cranial primary respiratory mechanism. There appears to be a higher incidence of category I pelvic fault correlation with sacral and cranial primary respiratory dysfunction.

The category I pelvic fault can adversely influence the cranium in ways other than through the dura and spinal column. With a category I fault there is nearly always shoulder girdle adaptation. This imbalance adversely affects the balance of the sternocleidomastoid and upper trapezius muscles, which pull into the cranium and can possibly create cranial faults. As we continue with discussion of the category I pelvic fault, remember that any structural imbalance below the cranium can adversely influence the cranial primary respiratory mechanism. This includes the feet, gait, modular balance, and many other factors.

Examination

The examination procedure is best done with the patient prone. The category I pelvic fault is characterized by positive therapy localization with one hand over each sacroiliac articulation. Therapy localization is most often evaluated by testing a previously strong indicator muscle for weakening. In the presence of this bilateral therapy localization, the fault is further evaluated by doing a two-handed therapy localization over one sacroiliac, placing one hand over the other directly over the articulation. One sacroiliac or the other will show this two-handed therapy localization, which is considered the positive side of the category I pelvic fault.

The pelvis is also evaluated with challenge for a category I fault. When this fault is present, the pelvis is twisted in such a manner that one innominate bone is situated with the ilium posterior, and the other with the ischium posterior. The challenge mechanism follows the basic rule for spinal involvement; that is, a previously strong indicator muscle will weaken when the structure is challenged in the direction in which correction is needed. Challenge requires a two-handed contact. One hand is placed on the posterior superior iliac spine, and the other is on the ischium of

the opposite side. Force in the amount of four to eight pounds is applied simultaneously at the contact points and then released, followed by a test of a previously strong indicator muscle. A weakened muscle is positive indication of a category I pelvic fault.

The patient's leg length will correlate with the misalignment of the pelvis, with two exceptions. One is an anatomical short leg, which is rare; the other is an atlas subluxation causing the short leg to become long when the patient changes from a supine to a prone position. When the posterior superior iliac spine is posterior and inferior, there will be a short leg on that side. The posterior ischium will be on the long-leg side. If there is no correlation and the patient does not have an anatomically short leg or an atlas subluxation, switching will probably be present. After its correction, the challenge and leg length should correlate.

Block Adjusting Technique

The pelvis is corrected with a modified DeJarnette block technique.^{2, 8} With the patient prone, the blocks are placed under the pelvis. Block placement uses gravity to put torsion into the pelvis toward correction. One block is placed under the anterior superior iliac spine of the posterior ischium side, the other under the opposite acetabulum and femur head. The blocks point toward each other, causing the patient's body weight to introduce torsion into the pelvis. The physician then uses a pumping adjustive thrust on the side opposite double-handed therapy localization. This bears repeating: correction is done on the side opposite that considered positive. The contact is either on the posterior superior iliac spine or on the ischium, which is posterior. Thus the contact will be on the ischium if the block on that side is under the ilium. If the block is under the acetabulum and femur head on the side to receive the thrust, the contact will be on the posterior superior iliac spine.

Correction of the pelvis generally requires approximately twenty repetitions of the physician's pumping action. An excellent method for determining the exact therapeutic effort required is to monitor tenderness at the junction of the 1st rib, clavicle, and sternum. Because of the pelvic torque in a category I, there is nearly always tenderness at this junction; this is due to the compensating twist of the shoulder girdle from the torque forced into it by the category I. After successful treatment of the category I, the tenderness at this junction will be significantly reduced. Often it is almost entirely eliminated; an

Examination and Treatment of Pelvic Dysfunction

acceptable reduction is 50%. After the therapeutic attempt, the pelvis should be re-evaluated with therapy localization, challenge, and leg length check to determine the effectiveness of the approach.

This has been a brief review of the category I pelvic fault. For a more complete discussion, see Volume I.

Muscle Involvement

The category I pelvic fault is often involved with muscular imbalance. Specifically, the abdominal muscles appear to be responsible for creating and perpetuating this fault on a respiratory basis. The relationship of these muscles to respiratory movement of the pelvis and the primary respiratory function was discussed earlier under "Pelvic Motion" (page 204). There are two divisions of both the external and internal oblique abdominal muscles and the transverse abdominal muscles. These provide rotational support for the pelvis. The rectus abdominis is primarily involved in flexion and extension of the pelvis, but it may also have some bearing on pelvic rotation if it is unilaterally weak.

Various vectors of challenge on the innominate bone can help determine what muscle is imbalanced and thus contributing to a category I pelvic fault. This is in addition to the individual abdominal muscle tests discussed in the muscle testing section of Volume I.

Chart 7—15 categorizes the probability or possibility of muscle weakness with challenge. Two types of challenge are listed, one for rotation and the other for flexion-extension. The lateral crest of the ilium is challenged for its external rotation, which

equates to an internal rotation of the posterior superior iliac spine. Since the pelvis reacts to challenge with a rebound mechanism, the crest of the ilium is external when there is a weakening of a previously strong indicator muscle after the crest has been challenged from the lateral to the medial aspect. This would be listed as an external iliac crest. If the innominate in general is being listed, the listing would be an internal posterior superior iliac spine (IN-PSIS); when the iliac crest moves externally, the posterior superior iliac spine moves medially. When this type of challenge is positive, the muscles to consider are those drawing the crest of the ilium medially. The iliac crest can challenge opposite to that described, in which case the listing of the crest would be internal and the posterior superior iliac spine would be external (EX-PSIS).

The posterior ischium challenge is for flexion and extension, and is the same as that usually done when evaluating a category I. The ischium is challenged from the posterior to the anterior aspect while the opposite posterior superior iliac spine is challenged from the posterior to the anterior aspect. Various vectors are not used in this challenge, but the information of the rotational challenge for the ilium correlates with the ischium information.

The chart is a guide to determine the probability of muscular weakness; it should be correlated with individual abdominal muscle tests described in Volume I. Whenever there is a recurrent category I, the abdominal muscles should be thoroughly evaluated and corrected with the usual applied kinesiology approaches for muscle weakness.

CATEGORY I ABDOMINAL MUSCLE WEAKNESS CHART

Comparing the information obtained from challenge of the pelvis with this chart gives probable and possible indication of which abdominal muscles are weak in the presence of a category I pelvic fault. Because of the thoracic respiratory correlation to a category I, correction of these muscles is important in maintaining pelvic correction.

Key: Strong positive challenge (previously strong muscle weakens significantly after challenge) means muscle may be involved as indicated.

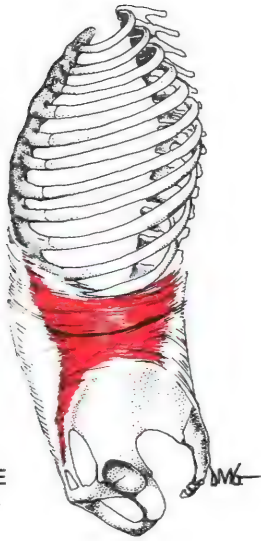
Muscle	PELVIC CHALLENGE	
	External Ilium	Posterior Ilium
External Oblique		
Anterior	Minor	Major
Lateral	Major	Minor
Internal Oblique		
Anterior	Major	O
Lateral	Major	O
Transverse Abdominal	Major	O
Rectus Abdominis		
Bilateral	O	Major
Unilateral	Minor	Major

Major = probably weak

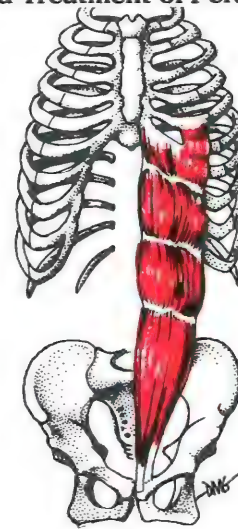
Minor = may be weak

O = probably not hypertonic or weak

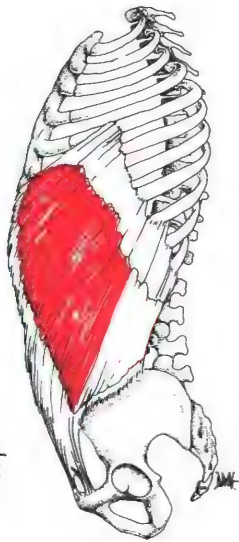
TRANSVERSE
ABDOMINAL



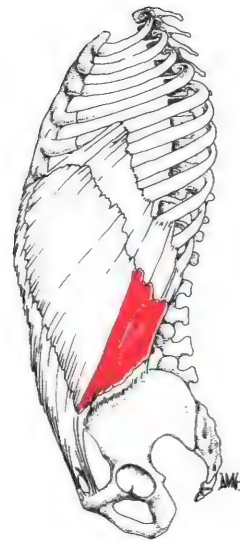
RECTUS
ABDOMINIS



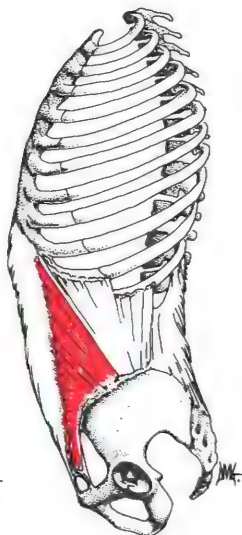
EXTERNAL
OBLIQUE
ABDOMINAL
(ANTERIOR
DIVISION)



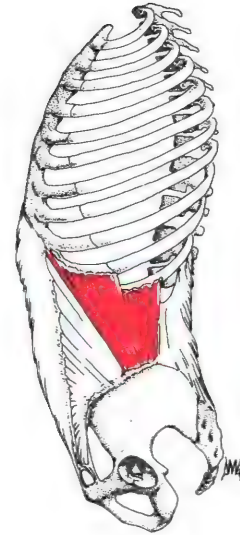
EXTERNAL
OBLIQUE
ABDOMINAL
(LATERAL
DIVISION)



INTERNAL
OBLIQUE
ABDOMINAL
(ANTERIOR
DIVISION)



INTERNAL
OBLIQUE
ABDOMINAL
(LATERAL
DIVISION)

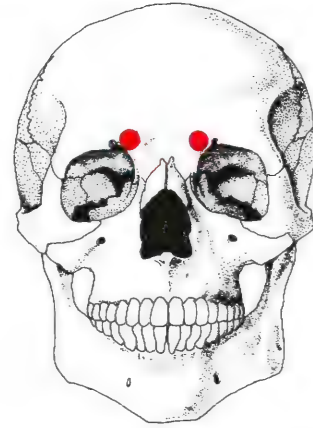


Examination and Treatment of Pelvic Dysfunction

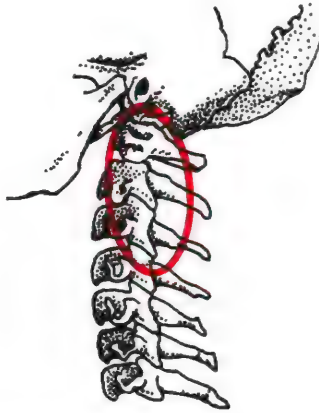
CRANIAL-SACRAL CORRELATION TO CLOACAL SYNCHRONIZATION TECHNIQUE

The cloacal synchronization technique in applied kinesiology appears to deal with intercommunication between some of the equilibrium proprioceptors. The method of examination for this involvement and its influence on health is presented in Volume I of this series of texts. There are two treatment methods for cloacal synchronization. The method dealing with cranial-pelvic function is not discussed in Volume I since the basic principles of the approach are presented here. First is a brief review, and then the cranial-pelvic corrective approach is presented.

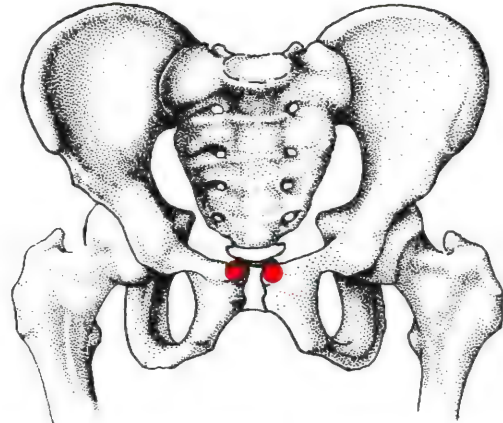
Indication for the necessity of this treatment is positive therapy localization over the reflex points — the visual righting, neck righting, labyrinthine, and anterior and posterior cloacal reflexes. In addition



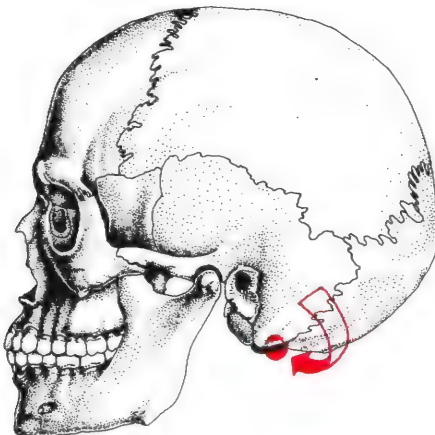
Therapy localization points — visual righting reflexes.



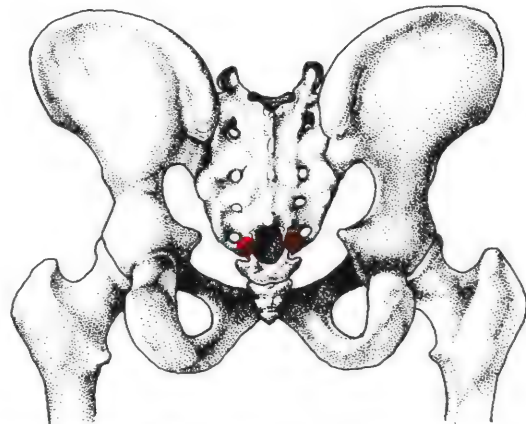
Therapy localization points — the neck righting reflexes.



Therapy localization points — anterior cloacal reflexes.



Therapy localization points — labyrinthine reflexes.



Therapy localization points — posterior cloacal reflexes.

there are muscle group tests, developed by Beardall,¹ of hip flexion and extension and shoulder flexion and extension; these are weak when two groups are tested together, but strong if each group is tested separately. (For a complete discussion of cloacal synchronization technique and the examination for its requirement, consult Volume I.)

When there is positive therapy localization or two-group muscle tests indicating a problem, the test is repeated with respiratory assist to determine if the positive indication is removed. For example, if therapy localization is positive over the anterior cloacal and visual righting reflexes, the patient is asked to take a deep inspiration; the indicator muscle is re-tested while inspiration is held. If the indicator muscle regains its strength with a phase of respiration, involvement of the cranial-sacral mechanism is indicated. The other examination procedure — two-group muscle tests — is also used to test for respiratory assist. Both therapy localization and the muscle tests should have the same phase of respiration assist, either inspiration or expiration. If there is no correlation, the patient is neurologically disorganized; evaluation for and correction of the disorganization should be done prior to proceeding. After the neurologic disorganization has been corrected, the two aspects of examination should correlate.

If there is a respiratory assist to cloacal synchroni-

zation examination, the preferred correction is cranial and pelvic treatment dealing with the primary respiratory mechanism. If there is no respiratory assist, use the reflex-electromagnetic therapy for cloacal synchronization described in Volume I.

The respiratory assist which eliminates the positive two-group muscle test or therapy localization indicates the type of cranial-pelvic treatment needed. For example, if the examination is positive for improper communication between the labyrinthine and posterior cloacal reflexes but negated by inspiration held in, inspiration assist or sphenobasilar inspiration assist on the side of involvement is indicated, or possibly sacral inspiration assist. It may be necessary to treat for both cranial and pelvic involvement.

This information indicates only that treatment is needed; it does not specifically determine the area to be treated. It is necessary to challenge and otherwise evaluate the cranium and sacrum for possible correction. If the cranial and pelvic involvement is ipsilateral, it is very likely that both will need treatment. If the involvement is contralateral, only the cranium or sacrum will usually need correction. If both contralateral areas show positive challenge, the patient is probably neurologically disorganized and should be evaluated and treated for that before further therapeutic efforts are attempted.

If the visual righting and anterior cloacal reflexes



7—18. *Internal iliac crest corrected with inspiration assist. Physician pulls anterior superior iliac crest laterally and posterior superior iliac crest medially with patient's inspiration. Challenge first to determine optimum vector.*

Examination and Treatment of Pelvic Dysfunction

are involved, the patient will have some type of cranial fault affecting the eye on that side. The fault most frequently found is an internal or external frontal rotation. Although this is the most common fault involved with the visual righting reflexes, it is not the only one which could be present. Because of the interrelationship of all the faults and the possibility of any cranial fault creating nerve entrapment, the skull should be evaluated to determine the type of cranial fault creating the problem, and then corrected.

The anterior cloacal reflex usually requires therapeutic pressure on the anterior superior iliac crest in a generally lateral direction because the crest moves laterally on inspiration; correction is done on a direct basis rather than by rebound. Evidence indicating the need for this lateral correction is positive therapy localization negated by an inspiration assist. Challenge the iliac crest laterally with various vectors to find the maximum weakening of a previously strong indicator muscle. This will be the appropriate vector of force to apply on inspiration. Repeat the pressure with four or five inspirations and re-evaluate. If the therapy localization or muscle test for the anterior cloacal was negated by an expiration, the anterior superior iliac spine is pressed medially on expiration. Again challenge to find the best vector of correction.

When there is poor communication between the visual righting and anterior cloacal reflexes, treat-

ment may be required at the cranium and pelvis in either ipsilateral or contralateral combinations. The same cautious approach to neurologic disorganization indicated in posterior involvement does not seem to be present in contralateral anterior conditions. This is apparently because improper innominate movement has an adverse influence on the anterior cloacal reflex, rather than the sacrum. Innominate respiratory movement is not as integrated with the cranium as sacral movement. As pointed out before, the innominate moves primarily with thoracic and diaphragmatic respiration, influencing the sacrum; however, it does not depend on sacral movement, which is specifically correlated with cranial movement. Of course, as with all other conditions, the physician should constantly be aware of the possibility of neurologic disorganization confusing the examination, but contralateral anterior involvement of the cloacal and visual righting reflexes does not specifically indicate it.

After correcting the cranial-pelvic factors of cloacal synchronization, the patient should be re-evaluated using therapy localization and the two-group muscle testing procedure. Correction of cloacal synchronization is very important in neurologic disorganization and general structural imbalance. It is also a great aid in helping maintain structural corrections which are obtained with other manipulative procedures.



7—19. External iliac crest corrected with expiration. Challenge to determine optimum direction of correction. Physician pushes laterally on the posterior superior iliac crest and medially on anterior superior iliac crest.

SACRAL WOBBLE

The sacral rotatory-type fault has been described by Goodheart as the "sacral wobble."⁶ This sacral involvement appears similar to the inspiration and expiration sacral faults already described, but it is more significantly locked and has more than the usual rotatory aspect. Evidence of a sacral wobble fault will sometimes be negated by category I or sacral treatment, without direct treatment to the fault itself.

Examination

The patient with a sacral wobble will have a characteristic gait in which it appears the sacrum is actually wobbling as the patient walks. Indication to test for a sacral wobble is when a muscle which tests weak (e.g., hamstrings) strengthens with a phase of respiration, but no sacral fault is found on challenge. The usual therapy localization over the sacrum will be present, causing a previously strong indicator muscle to weaken. To differentiate this weakening as a sacral involvement, have the patient take various phases of respiration while holding the therapy localization and re-test. For example, take a full inspiration and hold it while the muscle is re-tested. If the involvement is a sacral respiratory one, the indicator muscle will regain its strength with one of the phases of respiration.

The most significant difference between the

sacral wobble and standard inspiration and expiration sacral faults is the nature of the challenge. Challenge for a sacral fault is simply pressure on the apex of the sacrum in either an anterior or posterior direction, depending on respiratory assist. The sacral wobble will not show a positive challenge in this manner, but it will with a two-handed challenge. Challenge determines the side of treatment and differentiates the fault from other involvements.

To challenge for an inspiration assist sacral wobble, the physician contacts the lower one-third of the sacrum, basically over the apex on one side. With the other hand, the contralateral innominate is contacted on the anterior superior iliac spine. Challenge is by force applied with both hands, basically toward each other, so that the apex of the sacrum is moved anteriorly and medially; the anterior superior iliac spine is brought posteriorly and medially. The challenge is repeated with various vectors of force to find the maximum positive challenge. A positive challenge is a rebound one, causing a previously strong indicator muscle to weaken. The challenge is repeated on the opposite side, but it should be positive on only one side; this is the side of treatment.

To challenge an expiration assist sacral wobble, the physician contacts the anterior aspect of the sacral apex with a thumb; with the other hand he



7—20. Sacral wobble, inspiration assist. As the physician moves the sacral apex anteriorly and pulls the contralateral ilium posteriorly, the patient inspires and lifts the pelvis.

Examination and Treatment of Pelvic Dysfunction

contacts the sacral base with a pisiform contact. The challenge moves the sacral apex posteriorly while moving the contralateral base anteriorly. A positive challenge is indicated by a previously strong indicator muscle weakening. Again, the challenge should be repeated on both sides and with various vectors; only one side should be positive. The side considered to be involved is the one on which the apex challenge is positive. This is the case in both inspiration and expiration sacral wobble faults. The positive side is generally on the side of patient handedness. This is a general rule, not a specific one. Challenge must be done to determine the positive side.

Correction

Corrective effort is directed in the same vectors that caused the maximum challenge. In addition to the two-handed therapeutic pressure applied by the physician with the patient's respiration, the patient moves the pelvis in a specific direction with each phase of respiration.

The inspiration assist is corrected by moving the sacral apex anteriorly, while simultaneously pulling the anterior superior iliac crest in a posterior direction, indicated by the vector of challenge. Before starting the maneuver, the physician should instruct the patient regarding pelvic movement. On inspiration the patient is to move the pelvis posteriorly, thus basically lifting it away from the treatment table and pushing the sacrum more forcefully into the

doctor's treating hand. On expiration the patient moves the pelvis anteriorly, or toward the table. The therapeutic effort is repeated with four or five inspirations. The physician should feel the pelvis loosen and begin to yield when correction is obtained.

Correction of the expiration assist sacral wobble is also accomplished with the same contact and vector of force that produced the maximum challenge. As the patient exhales, the sacral apex is moved posteriorly and its contralateral base anteriorly. Instructions are given the patient to move the pelvis anteriorly toward the table on exhalation and lift it away from the table on inspiration. Again, motion of the pelvis will be felt when the therapeutic effort is effective.

After the corrective attempt is made on the sacral wobble, re-evaluate with therapy localization and challenge to confirm the correction. If correction was not obtained, repeat the procedure.

The sacral wobble is often associated with basic structural faults, and it may mask some of them. Prior to correction of the sacral wobble, the patient may be basically balanced on plumb line analysis; he may reveal many distortions after the correction. The sacral wobble correction is essential for holding many manipulative corrections.

The sacral wobble is often associated with an upper cervical cranial distortion called atlanto-occipital counter torque.



7—21. Sacral wobble, expiration assist. Patient first raises his pelvis. On expiration he lowers his pelvis as the physician raises (posterior) the apex and pushes down (anterior) on the contralateral base of the sacrum. Repeat until sacroiliac motion is felt.

ATLANTO-OCCIPITAL COUNTERTORQUE

The atlanto-occipital countertorque is of a primary respiratory nature and probably correlates with a torque of the dura from within the skull to the upper cervical region. This involvement is often associated with a sacral wobble fault. When it is found, the sacrum should be evaluated for inspiration or expiration assist sacral faults or sacral wobble.

Examination

The atlanto-occipital countertorque is best therapy localized by having the patient place one finger on the occipital bone, one on the mastoid process, and one each on the atlas and axis. As in other areas, therapy localization indicates only that something is involved; without other correlation, it is not pathognomonic of the atlanto-occipital fault. One step toward differentiation is to have the patient take either a deep inspiration or expiration; then determine if positive therapy localization is eliminated. If so, the presence of some type of respiratory assist fault is indicated. Challenge as indicated below will give final positive differentiation. Although this involvement will generally show positive therapy localization with fingers in only the general region, there are numerous cases that would be missed with poor therapy localization. It is easier to use a previously strong indicator muscle and evaluate for weakening, rather than attempting to find a muscle weak as a result of the fault and cause it to strengthen with therapy localization.

Challenge is accomplished with one contact on the atlas transverse process and the other on the mastoid process. An inspiration assist fault is challenged by pushing anteriorly on the posterior

aspect of the mastoid process, while the physician's other hand pushes posteriorly on the atlas transverse process. The challenge is released, and a previously strong indicator muscle is tested for weakening. As with other cranial challenges, various vectors of force are applied until the maximum positive challenge is found. This is the best direction for correction.

The opposite challenge is used for an atlanto-occipital expiration assist. The anterior aspect of the mastoid process is contacted and pushed posteriorly, while the transverse process of the atlas is pushed anteriorly. Three or four pounds of pressure are used on these challenges.

If a sacral wobble fault is present, it should correlate with the findings at the atlanto-occipital area. There should be an inspiration or an expiration assist ipsilaterally at both the cranium and sacrum. If not, the patient should be evaluated for neurologic disorganization and the condition corrected, if present. After that correction, the two involvements should be on the same side.

Correction

Treatment for the atlanto-occipital countertorque is done in the same direction as the optimum challenge. Pressure is applied four or five times with the same phase of respiration indicated in the examination.



7—22. One finger is on the occipital bone, one on the mastoid process, and one each on the atlas and axis for therapy localization of atlanto-occipital countertorque.



7—23. Inspiration assist above and expiration assist below.

Recurrent Cranial and Pelvic Faults

Ideally, correction of a cranial fault should be permanent. The usual approach is to evaluate immediately after correction to make certain it has been obtained, then re-evaluate the cranium on the patient's next office visit to determine if the correction has held. If the correction has not held there are usually two reasons for its failure: (1) the cranial dysfunction is very chronic, or (2) there are other involvements feeding dysfunction into the cranial-sacral primary respiratory mechanism, causing the fault to recur.

The chronic resistant cranial fault is usually recognized when the physician first begins to work with the mechanism. This fault is probably present in the skull which feels very rigid during challenge and therapeutic attempts. Some skulls are so rigid that correction achieved with the first therapeutic attempt is limited. The first few visits may show little motion gained. In this case, it is simply necessary to continue working with the mechanism to regain normal motion. Although in a few cases chronicity is capable of causing slow progress in the beginning, care must be taken that it is not blamed for lack of improvement when other problems are feeding stress into the system.

Other areas of dysfunction in the body are more common causes of recurrent cranial faults and general failure to obtain correction. The associated involvements may be closely related with the cranial primary respiratory mechanism. They may be located in the skull, cervical spine, or pelvis. On the other hand, the involvement may be remote and seemingly unassociated with the cranial-sacral primary respiratory mechanism.

The intrinsic muscles of the skull should be evaluated with every cranial fault. These muscles have been listed throughout the section on cranial correction. The muscles involved may directly cross a suture, or they may be somewhat more remotely involved by inserting into fascia that ultimately crosses the suture. The intrinsic skull muscles are usually hypertonic, creating a jammed suture. The muscles and cranium can be more thoroughly evaluated by having the patient contract the muscle(s) while therapy localization or challenge is done. An example is to have the patient raise his eyebrows as high as possible, contracting the frontal and probably occipital bellies of the occipitofrontalis muscle. This may cause a jamming of the lambdoidal or coronal suture which was not observed in the clear. Observation of wrinkles caused from persistent muscle contraction may give clues as to which muscles are

hypertonic. Involvement may be exhibited by an increased nasolabial fold, indicating the zygomatic muscles, or wrinkles at the glabella, indicating the procerus or corrugator supercilii. Proprioceptive treatment is usually the choice method for correction; however, spray and stretch or fascial release technique may be necessary.

Imbalance of the muscles of mastication, temporomandibular joint dysfunction, malocclusion, and pelvic faults are probably more involved with recurrent cranial faults than any other problems. It should be obvious that the very powerful muscles of mastication, originating from various cranial bones, put massive forces into the skull during the chewing process and, yes, even when talking. The mandibular condyles articulating with the mandibular fossae also place force into the skull. A malocclusion of the teeth may throw this force off-balance, requiring dental equilibration or bite plane therapy. (Evaluation and treatment of the muscles of mastication and the TMJ are discussed in the second section of this book.)

As has been mentioned many times previously in relating with the various cranial faults and mechanisms, there are many postural and spinal muscles which pull directly into the cranium. Some of the more important ones are the sternocleidomastoid, upper trapezius, longissimus capitis, semispinalis capitis, obliquus capitis superior, rectus capitis posterior (major and minor), and the longus capitis. Obtaining balance of these postural muscles may require local treatment, such as cervical spine manipulation. It may be necessary to organize the body modules to function on an improved basis with each other with PRYT technique, gait mechanism, cloacal synchronization, etc.

An imbalance of the muscles supporting the pelvis can initiate cranial dysfunction. Imbalance may be from the sacrum directly influencing the cranial-sacral primary respiratory mechanism. Failure to correct this imbalance will generally cause cranial faults to return as soon as the patient walks, perhaps even before. The muscles influencing the innominate bones, which in turn influence the sacrum, will also cause cranial faults to return on walking. The influence of abdominal muscles on the innominate bones may cause cranial faults to return simply with deep breathing.

The adverse effect on the cranial-sacral mechanism may be from remote structural problems. An example is a foot or knee subluxation improperly stimulating the proprioceptors and feeding erroneous information for facilitation and inhibition of muscles

directly responsible for stabilizing and moving the pelvis and cranium. As an individual walks there is facilitation and inhibition of many muscles, which have been described in this section, responsible for balance of the cranial-sacral primary respiratory mechanism. Correction of these remote subluxations must be accomplished for permanent cranial correction. The remote involvement may not entail a subluxation; an improperly functioning muscle proprioceptor may be feeding improper information into the neuronal pools.

The patient's habits or occupational patterns may be responsible for the re-creation of cranial faults. School children often prop their chins on hands while reading, which throws stress into the mandible, traversing into the cranium. Reading in bed and propping the head on a hand creates similar stress to the skull. Using the head to apply force to some object, such as a sheet rock worker holding a sheet against the ceiling while it is nailed into place, creates stress not only to the cranium but to the cervical spine as well. Articles attached to the head, such as a welder's helmet, may create additional stress in the cranium.

Personal habits — pipe chewing, chewing on a pencil, etc. — may create stress in the cranium through the temporomandibular articulation and muscles of mastication. Gum chewing appears to sometimes cause subclinical cranial faults; it also sometimes hides cranial faults which would be present in the clear. It is probable that many who have a habit of chewing gum on a regular basis do so in a subconscious effort to mobilize the skull. If a patient chews gum prior to and through a cranial evaluation, many cranial faults may be missed.

Primarily the structural side of the triad of health has been discussed here. There can also be causative factors from the chemical and psychological sides of the triad, which may be direct or indirect.

There is considerable clinical evidence that chemical imbalance can influence the cranial primary respiratory mechanism. The reason for this influence is unknown. An example of chemical influence that is often seen is hydrochloric acid and its apparent relationship with a temporal bulge cranial fault. All evidence of a temporal bulge may be present, such as therapy localization, bilateral pectoralis major (clavicular division) weakness, challenge, and half-breath-in respiratory assist. The patient may suck on a hydrochloric acid tablet and eliminate all indicators for the cranial fault, including the challenge. Removing the tablet from the mouth and having the patient suck on an antacid tablet returns all indicators immediately.

Another illustration of a similar situation is to

mechanically clear all indications of a temporal bulge cranial fault, evaluate the individual for a contralateral parietal descent and any pelvic involvement, and correct these, if present. Continued re-evaluation over many days reveals the cranium and pelvis continue to function normally, but as soon as the patient sucks on an antacid tablet all eliminated indicators of a temporal bulge fault return. These almost amazing reactions of the cranium to chemicals point out the importance of the physician being aware of the nutritional or chemical factors the patient is using. It is especially a problem when a patient is using hydrochloric acid supplementation, which abolishes all indicators of the cranial fault. The patient may be showing symptomatic improvement from the hydrochloric acid supplementation, but the basic underlying cause is not being corrected. Upon discontinuing the supplementation, the symptomatic pattern will return. The best approach is to be certain that the cranial-sacral primary respiratory mechanism is corrected, which usually eliminates the need for hydrochloric acid supplementation. Whenever a patient has a recurrent temporal bulge and the physician has conscientiously looked for contributing factors which cannot be found, he should suspect that the patient is taking antacids and failing to report their use. A positive comment to the patient such as, "As I evaluate your nervous system, it indicates that you're taking antacids. How many per day?" will gain a much more positive response from him than the simple question, "Are you taking antacids?" Often the patient is aware, or thinks, that the doctor does not approve of antacids. This is especially true when the physician involved deals primarily with natural health care. Often patients will take over-the-counter medications on the sly, not wanting the doctor to know of the action. The positive question, "How many are you taking?" usually obtains the truth, as opposed to the simple question, "Are you taking antacids?"

The examples of hydrochloric acid and antacids are the most common observed in working with the cranial-sacral mechanism. It is likely that many other nutritional factors and chemical compounds also influence the cranium, but they have not been as specifically documented.

The mental or psychological side of the triad of health can also perpetuate cranial faults. Because the facial muscles are muscles of expression and reflect the mental attitude of an individual, they are often contracted on a continuous basis, thus influencing the cranial mechanism. Often these muscular patterns can be improved by the already mentioned proprioceptive, spray and stretch, and fascial release techniques of applied kinesiology. The basic mental

Examination and Treatment of Pelvic Dysfunction

attitude of the patient may need changing to prevent recurrence.

As can be readily observed, many factors can adversely affect the cranial-sacral primary respiratory mechanism. The occasional problem patient may require the in-depth investigative effort of a true detective. Several factors which have been mentioned in this section may need evaluation in various ways. If the patient obtains temporary relief from your cranial therapeutics, you know you are on the right track. Even if relief lasts only an hour or two, it tells you that some factor is causing re-creation of the cranial faults. Try to determine what type of activity the patient was doing prior to recurrence of the symptoms. If there was considerable walking or running, evaluate for weight bearing, gait mechanism, foot problems, etc. If the symptoms developed immediately after a meal, evaluate the chemical aspects of the patient, digestive function, temporo-mandibular joint function, etc. Consistent symptomatic development after emotional encounters requires evaluation of that aspect of the patient's total health picture.

Nearly all cranial-sacral primary respiratory problems are brought under control with relative ease when accurate, proper procedures are followed. The unusual case that has recurrent involvement simply

requires further investigation to find what is adversely affecting the mechanism.

Classification of cranial faults into the fourteen considered here is an academic approach for learning cranial function. It must be realized that the skull motion is that of a closed kinematic chain. Motion — or lack of it — in one area may influence remote areas in the skull. This means that cranial faults are, in reality, a continuum; it is almost impossible to have a particular type of cranial fault and not have numerous other areas of the cranium affected. It becomes obvious, then, that the fourteen faults considered here only present the principles of motion, examination, and correction. Since the cranial mechanism is a closed kinematic chain and cranial faults are a continuum, the physician should be able to apply the basic principles presented here to use challenge and therapy localization, breathing procedures, and muscle testing as methods to examine the skull beyond the fourteen cranial faults. As expertise is gained, the variations that different individual cranial problems present will be easily handled. Even though it is rarely necessary to deviate from the fourteen cranial faults listed, it is helpful to have a working knowledge of the skull so that custom approaches to the unusual skull can be designed by the physician in charge.

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Section II

Stomatognathic Area

Chapter 8

Stomatognathic Area Introduction

Terminology

Section II of this text deals with the jaws and temporomandibular joints, referred to here as the stomatognathic area. In Section I the stomatognathic system is defined as including basically all structures from the shoulder girdle up. These structures are described as being very closely integrated with the rest of the body.

As you began reading this text, your interest may have been only in the temporomandibular joint or in cranial dysfunction, with the knowledge that the area can cause many different types of health problems. If you began the text with an understanding of only one area, it is hoped that further investigation of your interest will lead you to the other area. Study of the total mechanism reveals it is impossible to separate cranial and temporomandibular joint function. The stomatognathic area can potentially influence the cranial primary respiratory mechanism in either a positive or a negative way, and the converse is true; the cranial primary respiratory mechanism is capable of influencing the stomatognathic area in either a positive or a negative way. The two are inseparable entities.

Discussion in Section II is at first limited to the mouth, including the jaws and teeth, which is called the stomatognathic area. There is then an integration of this area with the other factors of the stomatognathic system as defined by Shore.⁶³ The hyoid mechanism as conceived in applied kinesiology

is discussed, followed by myofunctional therapy. There is some overlap in the discussion from area to area to enable the reader to deal with occlusion or the temporomandibular joint without constantly having to refer to another chapter for an understanding of subject matter which is not the major topic of the chapter being read.

Section III presents further discussion of the interaction within the stomatognathic system and its interaction with total body function. The final integration of body systems and functions is presented in Volumes IV and V.

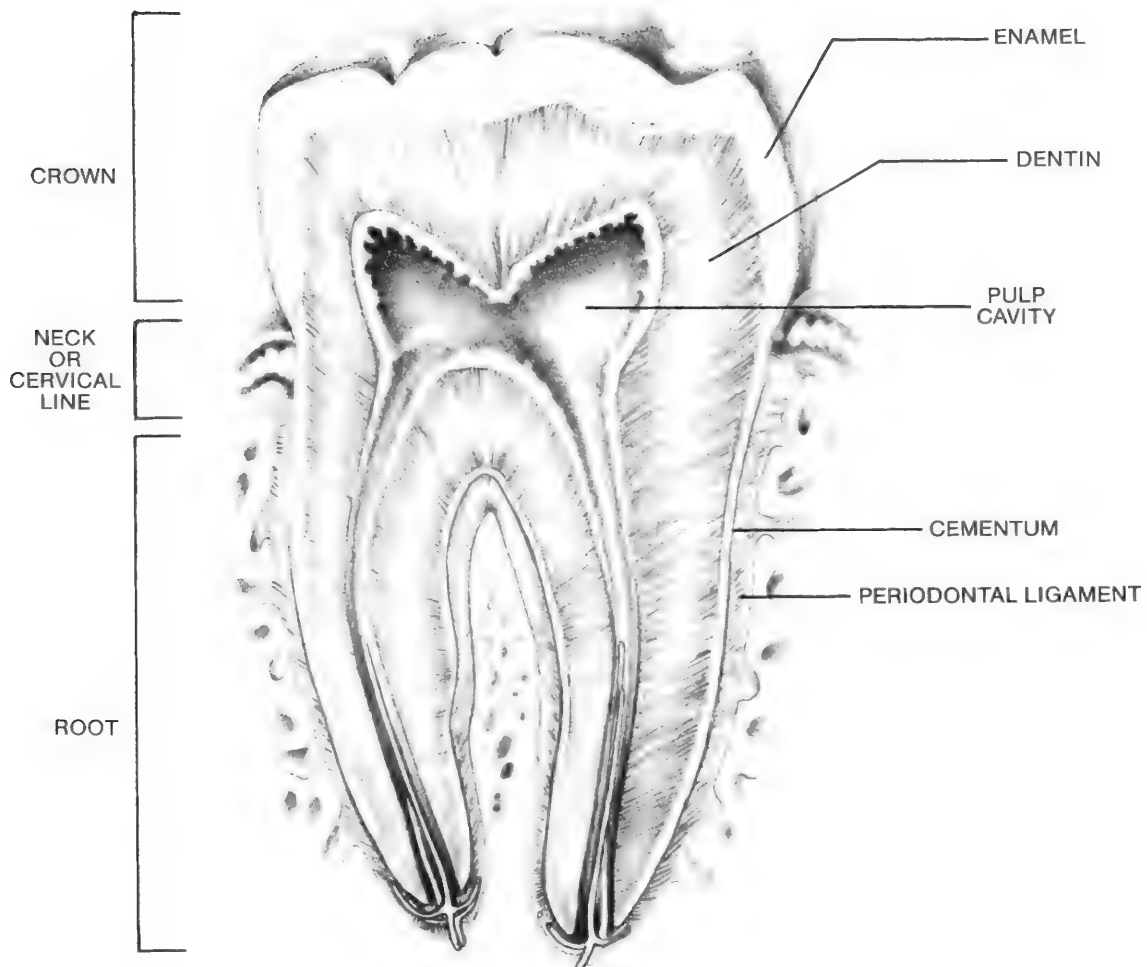
As we begin Section II, it is hoped the material will open new vistas of examination and treatment for many. It is unfortunate that more physicians are not aware of the major influence that the stomatognathic area and cranial primary respiratory mechanism have on health. Some who do not recognize that dysfunction in this area is significantly important to general body health denounce the approach, quoting minimal studies,^{28, 38, 41} while others actively promote the approach.^{25, 30, 41, 53} This presentation contends that there is a wide range of influence on the body, and also that many areas of remote dysfunction can adversely affect these structures.

It is best to begin this section with dental terminology,^{48, 49, 77} since many who read this text are not familiar with it.

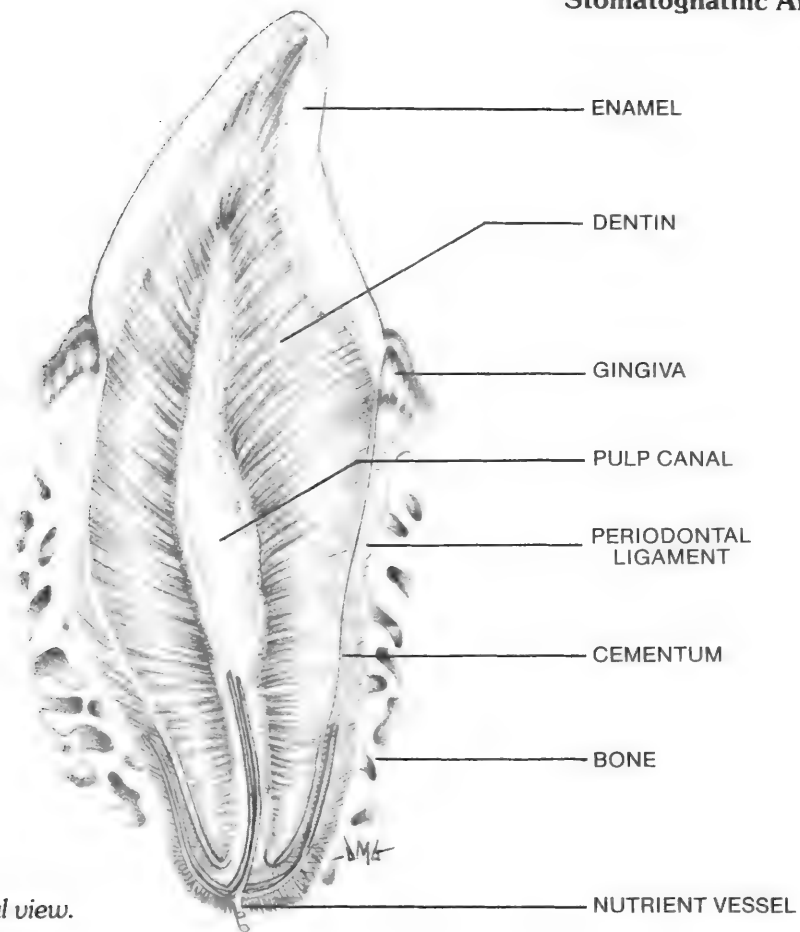
Teeth

A tooth consists of the crown and the root portions, divided by the cervical line. A cross section of a tooth reveals its four tissues — enamel, cementum, dentin, and pulp. The pulp is soft tissue furnishing blood and nerve supply to the tooth, while the other three are hard tissues. The crown has a

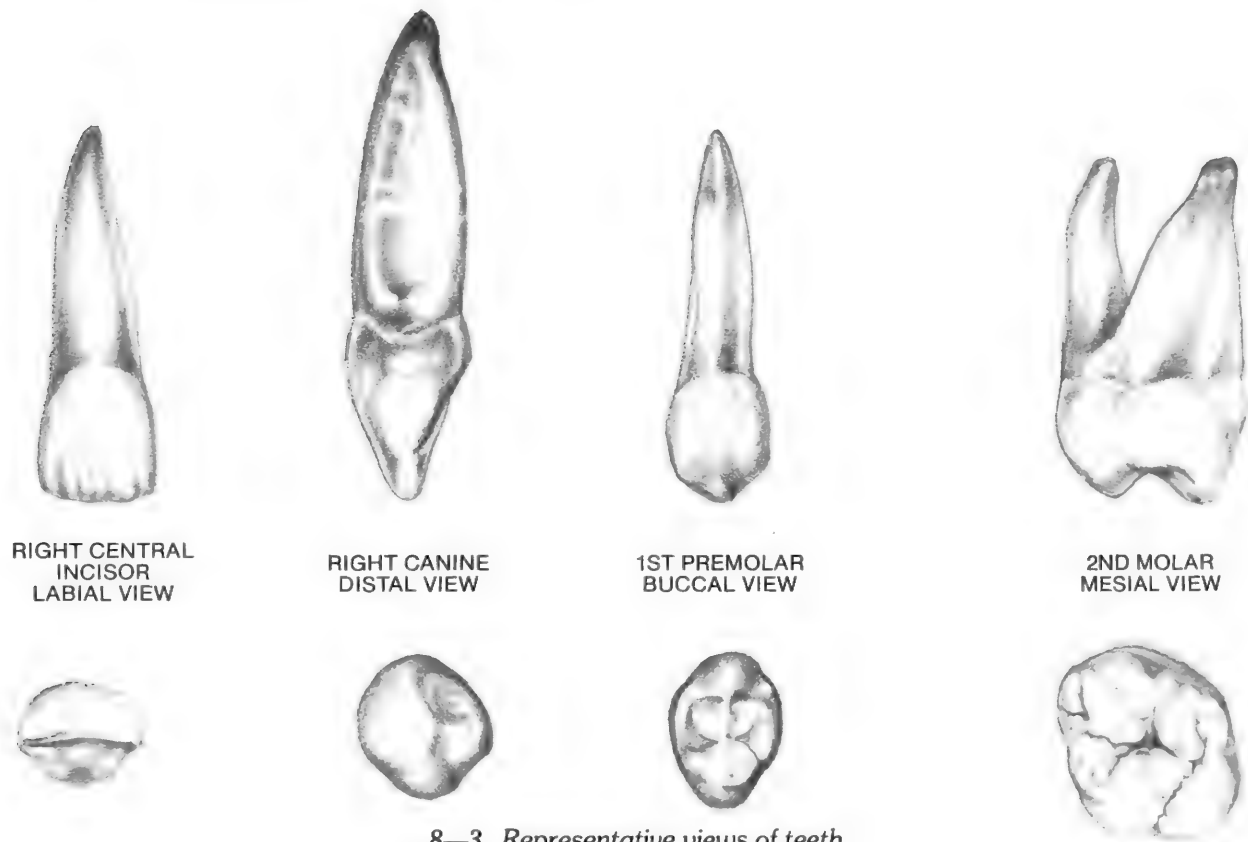
cutting edge which, in the case of the incisors, is an incisal ridge; for the rest of the teeth there are cusps. A cusp may be singular, as in the canine teeth, or there may be two or more cusps as in the pre-molars and molars. Intercuspalation occurs when the cusps of the teeth of the upper and lower jaws come together.



8—1. 1st molar tooth — buccolingual view.



8—2. Incisor — sagittal view.

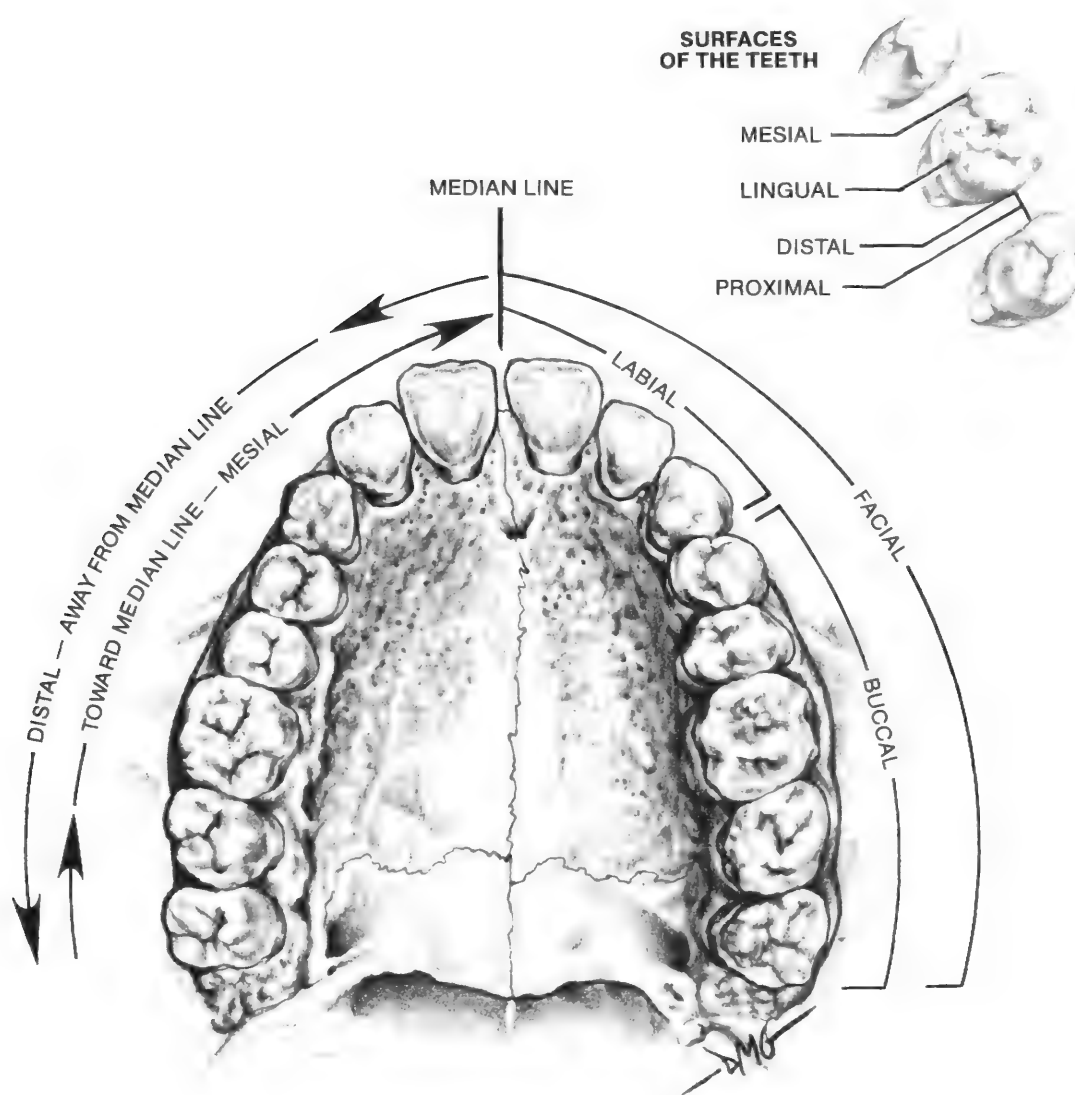


8—3. Representative views of teeth.

SURFACES OF TEETH

The crowns of the incisors and canines have a ridge which is the cutting surface, plus four other surfaces. The pre-molars and molars have the occlusal surface as their cutting surface, along with four other surfaces. All surfaces which face the tongue are lingual surfaces. The outer surface of the pre-molars and molars is called the buccal surface, and the outer surface of the incisors and canines is called the labial surface. The outer surface of the

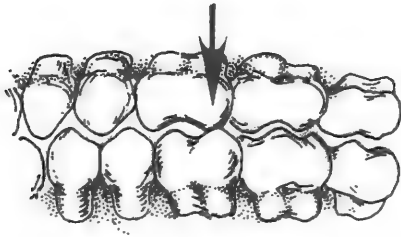
entire arch is referred to as the facial surface. The surfaces of the crowns which face adjoining teeth in the dental arch are called proximal surfaces. They are further distinguished as mesial or distal, which refers to direction in the dental arch. The proximal surfaces facing the middle of the arch are called mesial surfaces, while those facing the extremes are called distal surfaces.



GENERAL TERMS

When the teeth of the mandibular and maxillary arches come into intercuspation in a functional arrangement, it is called “centric” or “central occlusion” (CO). When this intercuspation is abnormal, the result is malocclusion. The term “centric relation” (CR) refers to the condyles of the mandible in their functional position in the mandibular fossae. There is considerable discrepancy in the literature regarding the definition of centric relation.

Prematurity refers to a first tooth contact which deviates the mandible from centric relation prior to complete intercuspation, producing a “slide” which is part of malocclusion. Most authorities state that centric relation must be in harmony with centric occlusion.

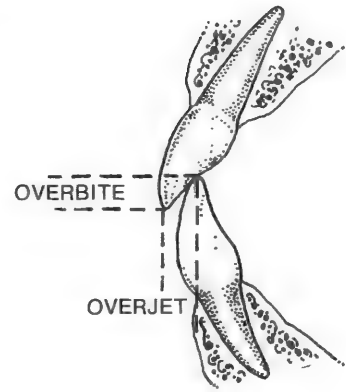


8—5. Exaggerated illustration of a prematurity contacting before the other teeth.

A crossbite occlusion is where the opposing upper and lower teeth are reversed from the usual occlusion. This places the lower posterior or incisor teeth outside the upper dental arch. There may be an anterior or a posterior crossbite. When posterior, it may be uni- or bilateral.

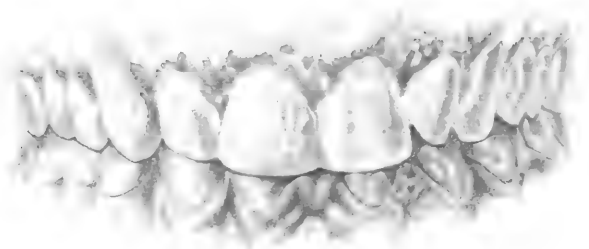
Overbite and overjet refer to the relation of the central incisors when the teeth are in centric occlusion. Normally, the incisal edges of the mandibular incisors occlude against the lingual surface of the maxillary incisors in such a way that approximately one-third of the mandibular incisor crown is overlapped by the upper incisor. This vertical overlap is called overbite. The projection of the upper incisors beyond their antagonists in a horizontal direction is called overjet, sometimes referred to as horizontal overlap.¹⁷

Closed bite refers to excessive overbite where little or none of the lower incisors are visible during centric occlusion. Open bite refers to failure of some teeth to contact when in centric occlusion.

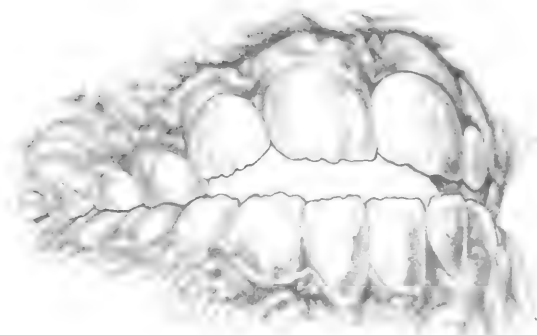


8—6. Overbite is a vertical dimension; overjet is a horizontal dimension.

The term “freeway space” or “interocclusal distance” refers to the distance between the occluding surfaces of the maxillary and mandibular teeth when the mandible is in its physiologic rest position. The “physiologic rest position” is the position of the mandible when the muscles of mastication are inactive. They have no electrical activity as observed on electromyography.



8—7. Closed bite.



8—8. Open bite.

ORTHODONTIC NOMENCLATURE

Mayoral^{148, 49} defines standard orthodontic nomenclature to describe the anomalies of position, direction, size, and shape of the jaws, lips, and other structures of facial and oral morphology. Greek and Latin roots are used to designate the variations of different places. Some of this nomenclature is presented here.

Place or Seat of the Anomaly

cheilos, lip
stoma, mouth
gnathos, jaw
geneion, chin
gonia, angle (of the mandible)
odontos, teeth
condyle, part of the mandible
occlusion, relation between the teeth of the upper and lower arches

Roots to Designate the Nature or Class of the Deviation

Changes of Position and Direction of the Soft Parts, Jaws, and Temporomandibular Joints

pro, in front
retro, back
dextro, right side
levo, left side
supra, above
infra, below

Changes of Position and Direction of the Teeth

gression, to walk, step, move (change of position)

version, to turn (change of direction, turning around a horizontal axis)

rotation, wheel (change of direction, turning around a vertical axis)

linguo, toward the tongue

labio or *bucco*, toward the lips or cheek

mesio, toward the middle of the dental arch

disto, toward the extremes of the dental arch

in, in or into

e (ex), outside, out of

Changes of Occlusion

linguo, toward the tongue

labio or *bucco*, toward the lips or cheek

mesio, toward the middle of the dental arch

disto, toward the extremes of the dental arch

hyper, excess

hypo, defect, less

Combination of these roots shows the structural deviation; for example, prognathism means that the jaw is in front of the place considered normal. Retrocondylism means that the condyle is behind the place considered normal.

This system of nomenclature deals primarily with orthodontic classifications, but can be applied to other uses. An example of accurate use of terminology can be given by evaluating the words "progression" and "protrusion." "Gression," indicating walk-step-move (change of position), infers the forward position of a structure. Compare this to "protrusion" where the root "trudere" means to impel with force, such as "the state of being thrust forward or laterally as in masticatory movement of the mandible."¹⁶

ANGLE CLASSIFICATIONS

Dr. Edward H. Angle (1855-1930)³ developed a system of classification of malocclusion. The classification is used extensively today, although it is a static one which does not take into account the dynamics of dental function. Relation of the mandible to the maxilla is based on the position of the maxillary first permanent molar.

Class I

Malocclusion characterized by normal mesio-distal relationship between the mandible and maxilla. The mesiobuccal cusp of the maxillary first permanent molar articulates into the mesiobuccal groove of



8—9. Mesiobuccal cusp of upper first molar articulating into mesiobuccal groove of lower first molar.

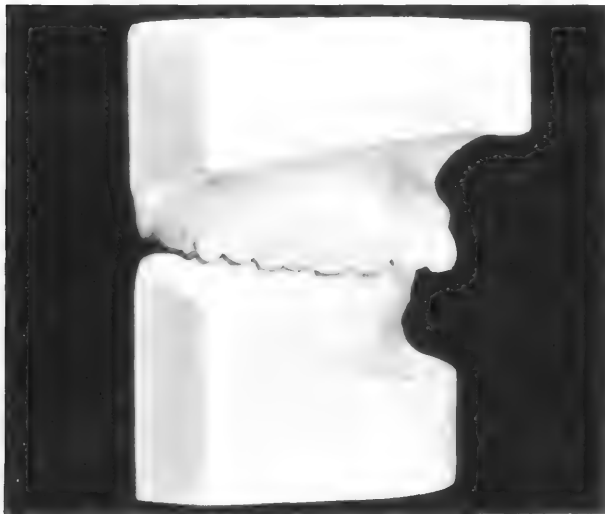
the mandibular first permanent molar. Malocclusion occurs in the anterior segments; one or many teeth may be deflected from their normal course.

Class II

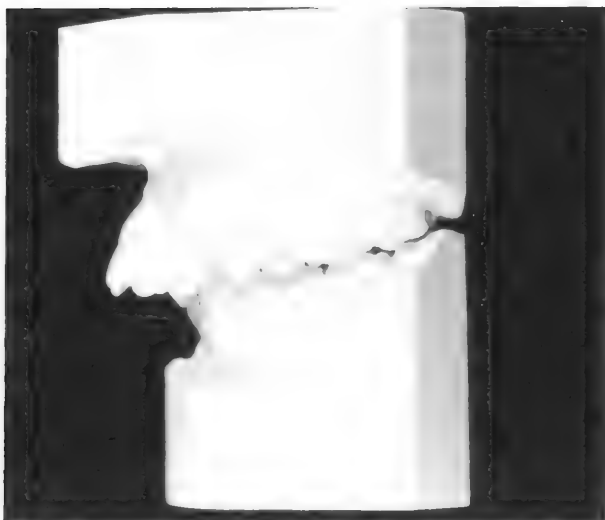
Malocclusion where there is regression of the mandible. The lower dental arch is distal to the upper on one or both lateral halves. The mesiobuccal groove of the mandibular first permanent molar articulates posteriorly to the mesiobuccal cusp of the maxillary first permanent molar.

Division 1: Bilaterally distal, with the maxillary incisors in labioversion.

Division 2: Bilateral distocclusion with retrogression of the maxillary central incisors. The maxillary lateral incisors tip labially and mesially.



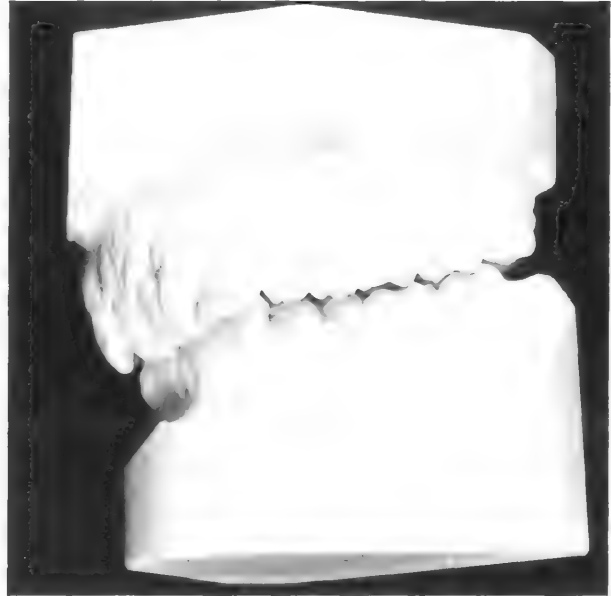
8—10. *Class I.*



8—11. *Class II, Division 1.*

Class III

Progression of the mandible with a mesial relationship of the mandible to the maxilla. The mesiobuccal groove of the mandibular first permanent molar articulates anterior to the mesiobuccal cusp of the maxillary first molar.



8—12. *Class II, Division 2.*



8—13. *Class III.*

The Stomatognathic System's Role in General Health Problems

In 1934 Costen,¹² a medical doctor, reported that headache and ear symptoms often result from disturbed function of the temporomandibular joint. His report received enough attention for the pattern of involvement to become known as Costen's Syndrome. The mechanisms which Costen related as causing the symptoms have been discredited.^{60, 61, 62} Although his mechanisms may have been incorrect, it seems apparent today that many of the conditions which Costen related to the temporomandibular joint are in fact caused by dysfunction in that mechanism and in surrounding tissues. Various theories have replaced Costen's. Dechaume¹⁵ described a major cause of the symptoms of temporomandibular joint dysfunction to be a result of intervention of the sympathetic system. This relates to the high concentration of terminal vascular nerves in the articulation being irritated. In addition to Costen's overclosure theory and Dechaume's neurologic irritation theory, there have been several who have proposed other theories, all of which are currently being practiced in varying degrees.

The tooth theory primarily deals with malocclusion causing displacement of the mandible. The tooth-muscle theory also relates with malocclusion but brings into the picture the neuromuscular system disturbed by stimulation to the periodontal ligament proprioceptors. The psychophysiologic theory primarily relates the myofascial pain syndrome to psychological causes that increase muscle tension.⁴³

Continued examination and research persistence have revealed that involvements in this area relate with many etiologies, encompassing nearly all the specialties in the healing arts. All specialties within dentistry are involved; the general dentist, prosthodontist, orthodontist, periodontist, and others — including the specialists of the temporomandibular joint itself — have specific areas relating uniquely to them. The internist and the orthopedist may be involved with systemic conditions, such as rheumatoid arthritis, infections, etc. The chiropractor has his role in structural balance and subluxations in the cervical spine. All of this, and we haven't even left the stomatognathic system to discover remote factors in the body which can influence this area! Not only can remote areas be influential, but we will see how the stomatognathic system may adversely influence health almost anywhere within the body. This wide range of influence from the area, and influence on the area, creates what could almost be considered as a "no man's land." An effort to write on this subject, in which so many specialties are involved, leaves one with great trepidation about stumbling into an area

not included in his province. Nevertheless, it is necessary to develop a stronger exchange of thought on an interprofessional basis since there is no one profession with the expertise to treat all the conditions which can cause disturbance in this system.

Even the terminology used to describe problems in this area indicates the diverse expertise necessary to evaluate and correct a condition. Distress patterns have been referred to as the temporomandibular joint syndrome (TMJS), myofascial pain-dysfunction (MPD), dental distress syndrome, masticatory myalgia syndrome, psychophysiologic theory, as well as many other names. Treatment approaches have been called cervico-oro-facial orthopedics and/or dental orthopedics, among other names.

The discussion in this text primarily reflects health problems resulting from interaction within the total stomatognathic system. This does not discount remote health problems which may develop as a result of localized disease processes in the dentition. Ratner⁵⁶ describes severe arm pain which can develop as a result of a pathological bone cavity in the alveolar bone of the maxilla. He relates to the work of Black and his colleagues^{2, 10, 76} who suggest a cause-and-effect relationship between alteration of the dentition and changes in the nervous system.

It is apparent that certain conditions, such as pain in the TMJ and muscles of mastication, may relate to temporomandibular joint dysfunction. In a study of 742 patients with disturbance in this area, Gelb²⁹ lists 320 (43%) as having pain in the temporomandibular joint. The next most common symptom was tinnitus (311, or 42%), followed by auricular pain (263, or 35%), headaches (152, or 20%), and bruxism (147, or 20%). In addition, there were various head pains and ear and temporomandibular joint symptoms.

The pain pattern of patients with temporomandibular joint dysfunction was charted by Perry.⁵⁴ Cases studied presented problems involving occlusion, but all conditions which could have developed as a result of severe traumatic injuries to the jaws or teeth were excluded from the report. Illustration 8—14 is a composite of the pain areas demonstrated by these patients. The dark areas were the ones most often reported; the lighter areas were reported less frequently. Generally the pain was unilateral; all areas represented in the composite were not demonstrated in every patient. Evidence in this study is quite strong that the pain was actually caused by dysfunction in the stomatognathic system. Tension in the system was evidenced by electromyography of the muscles of mastication, revealing activity even



8—14. Pain patterns with TMJ dysfunction as charted by Perry.⁵⁴

when the muscles were at rest. This abnormal activity subsided after occlusal therapy.

A common finding among investigators is headaches with cervical and masticatory muscle pain and tenderness. In one study of TMJ patients, Greene et al.³⁵ found that 81% of the patients evaluated had masticatory and cervical muscle tenderness. Of this group, the most common site of muscle pain was the external pterygoid muscle (84%); 43% of the patients had pain in the cervical, scalp or facial areas.

It seems easy to relate temporomandibular joint pain and headaches to TMJ malfunction. Dawson¹⁴ states that his personal clinical experience led him to believe that more than half of all chronic headache sufferers could be relieved of their pain by dentists. At first he was hesitant to accept responsibility for the relief headache patients experienced after an occlusion was adjusted, but continued favorable experience seemed to justify it.

Additional evidence of a probable correlation between headaches and TMJ dysfunction is presented in a study by Rieder.⁵⁷ Subjects (N = 153) from an apparently healthy population were questioned about

headaches, neck aches, and occlusal habits. Approximately half of the sample population experienced headaches and/or neck aches, and 42% indicated some type of occlusal symptom. A comparison of the group with pain and the one with occlusal symptoms revealed that 35% had an interrelationship of simultaneous symptoms. This study does not relate to an exact cause-effect relationship, but it does indicate that a very high percentage of "normal people" have headaches; a large number also have occlusal problems of some type. There was a higher number of occlusal problems in those who experienced headaches or neck aches.

The correlation of temporomandibular joint disturbance with the surrounding structures is evidenced by the wide range of tenderness found when an individual is examined for TMJ pain. Not only is the TMJ tender, but generally the muscles of mastication — the masseter, internal and external pterygoids, and temporalis — are also tender. There is also a high percentage of patients with pain in the posterior cervical, upper trapezius, and sternocleidomastoid muscles.⁶¹

What about the more remote involvements associated by some with temporomandibular joint dysfunction? In a study similar to Gelb's — but one which encompassed a wider symptomatic pattern of the body — Fonder²⁵ studied 247 patients with temporomandibular joint problems and found a higher percentage of involvement in remote areas. In his study all subjects had "... crepitation, subluxation, and a disturbance in opening and closing movements of the mandible. Every case was an easily recognizable condition of malocclusion. No questionable cases were used in this study. There was severe overbite; or several missing, overerupted, or tilted teeth, often accompanied by periodontal destruction; or a severe crossbite with missing and drifted teeth; or an excessive freeway space with an irregular occlusal plane." This study appears to have been more selective in the group evaluated. There were 92% of the cases with tinnitus and 73% with headaches. In addition, Fonder relates a high percentage of various symptoms including respiratory, ocular, visceral, and gynecological problems. He also relates mental symptoms, body postural problems, skin and hair symptoms, and general symptoms such as fatigue. The stomatognathic system as a possible causative factor of these symptoms may seem to many to be very remote. Fonder points out that malocclusion was not the only stress operable in the total person, and proper occlusion is not the panacea for all ills. Patients included in the study received therapeutics from many sources besides dentistry. He does maintain

Stomatognathic Area Introduction

that malocclusion is a contributing factor in a wide range of health problems. Applied kinesiology examination of the stomatognathic system and its relationship to total body function concurs with this belief.

There are established neurologic pathways which explain the pain of headache remote from the primary site of involvement in the stomatognathic area.^{7, 13} The interrelationship of headaches with the stomatognathic area, as well as other health problems, is discussed by Smith.⁶⁵ In addition to correcting an occlusion, he points out the necessity of consultation with other specialists in various health fields. From the opposite perspective, Berkman⁸ points out the necessity in some cases of correcting occlusion and temporomandibular joint dysfunction to help maintain chiropractic corrections. The interrelationship of the stomatognathic area with general body function and balance is also emphasized by Henningsen.^{39, 40} He discusses the interaction of the cranial primary respiratory mechanism with TMJ function, as well as integrating these areas with the rest of the body. The numerous interdisciplinary treatment approaches which are effective in this area are demonstrated in Gelb's book,³⁰ which covers a broad spectrum. Included are chapters on applied kinesiology, the cranial primary respiratory mechanism, myofunctional therapy, biofeedback, surgery, hypnosis, psychotherapy, occlusal therapy, muscular treatment, and endocrine disorders.

Although not the first, credit should be given to Costen¹² for making popular the observation that there may be auditory disturbances as a result of temporomandibular joint dysfunction. Although the mechanisms are viewed differently now, the association is observed by some in the otolaryngology field.

Arlen⁴ describes embryologic development, stressing the common origin of structures of the ear and masticatory system as an explanation for the clinical findings in this area. Symptoms of fullness in the ear, tinnitus, dizziness, and hearing loss are nearly always unilateral with what he calls the otomandibular syndrome. Patients describe the pain as dull and have difficulty locating it. It may be described as radiating into the neck, especially the sternocleidomastoid, and into the temporal region. There may be pain in the mastoid area, posterior cervical region, and down into the shoulder. These non-specific symptoms associated with ear pain are very different from frank ear disease, such as otitis externa or otitis media, in which the patient specifically locates the pain. Correction of the masticatory system returns normal hearing, which can be observed by improvement in audiogram and tympanogram test results. Many others have observed the correlation of

hearing problems with TMJ dysfunction.^{25, 27, 29, 47, 62}

It is relatively easy to understand that TMJ pain, headaches, neck pain, and hearing problems may be associated. But what about the claims made for improvement of conditions throughout the body as a result of TMJ treatment? Disturbance in the temporomandibular joint has been called "the great imposter,"⁵² and certainly many physicians seem to find that temporomandibular joint dysfunction can create problems throughout the body.

Possibly the greatest light was shed on the influence of the stomatognathic system on the total body by Tinbergen,^{24, 72, 73} the 1973 Nobel prize winner for physiology or medicine. He stated, "Many of us have been surprised at the unconventional decision of the Nobel Foundation to award this year's prize for physiology or medicine to three men who had until recently been regarded as 'mere animal watchers.'" Tinbergen used his skills in ethology (the scientific study of animal behavior) to contribute to the relief of human suffering. Familiar with the Alexander technique,^{1, 6, 74} he applied its principles to further study disorganization of body function. One of Alexander's discoveries was the importance of head-on-neck and neck-on-body balance. Alexander's original career was reciting dramatic and humorous pieces, but he developed serious vocal problems which nearly resulted in the loss of his voice. Since no doctors were able to help him, he began using the basic principles of ethology and studied himself. He found that a change of head-on-body position caused him to be unable to speak properly. Correcting this misuse of the body returned his voice to normal. Further study revealed that body function is adversely affected by many factors of improper use. Alexander continued perfecting his skill of observation and began teaching others proper body use. The Alexander technique and its wide range of influence on body function are discussed by Barlow.⁶

Alexander's original finding on himself, concerning the importance of the head-on-neck relation, appears to have great significance in understanding the integration of the nervous system. The muscles involved are very important in the stomatognathic system as described earlier in this text. As Alexander observed, there is great influence on the entire nervous system from dysfunction in this area. It can adversely affect the body by structural, chemical, or mental means.

There are many ways the factors Alexander and Tinbergen observed can cause dysfunction in the entire body by disturbance in the stomatognathic system. One easily recognized factor is the way that muscular imbalance which develops from occlusal

disturbances can ultimately involve the whole body. First it is important to understand that the jaw-closing muscles of mastication are part of a closed kinematic chain, and contribute to the chain from the mandible to the skull. The mandible, in turn, is anchored to the shoulder girdle by the supra- and infrahyoid muscles. The neck and head extensor muscles contribute to the posterior portion of the chain. Muscle hypo- or hypertonicity in any link of this chain disturbs its balance, and a vicious circle may develop. It may start as a result of TMJ disturbance which causes muscle contraction, and as Kraus⁴² states, “. . . pain of muscular contraction produces further muscle contraction over the short reflex arc and this contraction in turn increases pain and furthers more contraction, producing a vicious circle.”

This inappropriate muscle contraction causes reactions in remote areas of the body by way of stimulating the muscle's neuromuscular spindle cell and, depending on the location of the muscle, possibly equilibrium proprioceptors; the vicious circle begins. Information about the inappropriate muscle contraction is sent by the afferent system, which causes reaction in another muscle. The reaction becomes an action which sends information to cause an additional reaction. The far-ranging effects of the disturbance in the closed kinematic chain can go on and on.

Pain in the neck and shoulders associated with temporomandibular joint disturbance may be thought of as reflex pain by some; actually, there is abnormal muscle contraction in the neck and posterior shoulder, observed by electromyography, when the subject is at rest. Muscle activity at rest may be eliminated after occlusal correction.⁵⁴

Not only are the equilibrium proprioceptors of the cervical spine and head balancing muscles involved; as Goodheart³⁴ points out, the hyoid muscles are very important in the equilibrium and “centering” of the body. He compares the hyoid and its associated muscles to a gyroscope in the body (see Chapter 13).

An example of the wide range of influence is seen in Fonder's study²⁵ of the stomatognathic system's influence on the spinal column. In individuals with various types of malocclusion, a “removable template” was placed in the mouth to establish a physiologically balanced occlusion, and dramatic changes were made in spinal balance. X-rays prior to the occlusal change showed significant curves of the spine and general structural imbalance. X-rays taken four days after the occlusal change showed improved balance (see pages 4 and 5).

Mestman⁵⁰ presented a movie at the 1981

Summer Meeting of the International College of Applied Kinesiology which dramatically showed how occlusal change influences structural balance. The subject had head tilt and shoulder imbalance which were immediately balanced after change of the occlusion with a dental splint. Another subject had postural distortion and an aberrant gait; they were greatly improved when wearing the splint. Removal of the splint caused body distortions to return immediately, and the gait to again be disturbed.

It seems apparent that disturbance in the stomatognathic system can influence structure throughout the body. The question should arise as to whether malocclusion is the primary cause of disturbance in the stomatognathic system, or if sometimes it is secondary. Mintz,⁵¹ referring to “The Orthopedic Influence,” points out that the TMJ syndrome can be caused by postural imbalance. A short leg or a pelvic disturbance can create secondary imbalance(s) in the stomatognathic system. The influence may be to the neck and head extensors — one area of the closed kinematic chain — ultimately causing imbalance in the temporomandibular joint. It makes no difference whether the initial imbalance in the stomatognathic system is the posterior cervical muscles, hyoid muscles, or muscles of mastication; ultimately all will probably be involved.

Strachan and Robinson,⁷⁰ at the Chicago College of Osteopathy, were the first to observe a short leg's influence on malocclusion. Evaluating the pattern of masticatory muscles by electromyography, they removed a three-eighths inch heel lift from a standing subject's shoe and found an altered firing sequence of the muscles of mastication during chewing. When the lift was worn, the muscles showed the firing pattern of normal occlusion; with it removed, the firing pattern was one of a severe malocclusion.

General structural balance of the body is very important as a first step in correcting dysfunction of the stomatognathic system. Rogers,⁵⁸ a pioneer in the relationship of malocclusion to muscles, stated, “. . . our attention on the child naturally include(s) in our plans the correction of all postural defects. . . . The healing powers of nature rest upon balance and the restoration of balance, and whenever and wherever mankind has interfered with that balance he has paid the penalty.” Total structural balancing is a priority in the applied kinesiology approach to the stomatognathic system.

The interrelationship of the stomatognathic system with the rest of the body does not have to correlate with jaw function and occlusion. In some cases the only evidence of trouble is pain. Stoll⁶⁹ considered the many sources of innervation to the tongue. Cranial nerves V, VII, IX, and X are all

Stomatognathic Area Introduction

involved in some way. He pointed out how the tongue can become the seat of referred pain from a distant source of irritation in any organ innervated by the trigeminal, facial, vagus, or glossopharyngeal nerve.

Integrating these thoughts with the tremendous influence of the stomatognathic system as presented throughout this section of the text, we see a wide range of potential symptoms. The primary cause may be remote from the site of the symptom. This is true whether the direction of involvement is from the stomatognathic system to remote areas, or from remote areas to the stomatognathic system.

A procedure for evaluating circulatory function throughout the body and its possible association with the stomatognathic system is presented by Smith.^{66,67} By using plethysmography on the digits of the feet and hands and by Doppler ultrasonic evaluation of arterial flow, he demonstrated improved circulation after occlusal adjustment with a "wax bite" or with cotton roll supports. By thus changing the mandible's position, improved circulation was demonstrated on both the arterial flow and the capillary flow, indicated by the Doppler and the plethysmograph respectively. Further improvement in circulation is observed in his report after referral for osteopathic manipulation throughout the spinal column.

Influence of the stomatognathic system on general body function can be seen by its influence on muscle function as observed by manual muscle testing. This was originally demonstrated clinically by Goodheart.³³ These procedures have been demonstrated to the dental profession by Eversaul and Goodheart.^{19, 20, 21, 32} Many in the dental profession have used applied kinesiology procedures to evaluate the stomatognathic system. Some have done a very credible job of applying this system in the dental field. It must be pointed out that it is necessary to have a good working knowledge of manual muscle testing; a great number of errors can potentially develop as a result of its improper use. Failure to observe for substitution of synergistic muscles, patient movement, and improperly applied force can lead to errors and inconclusive findings.²⁸

Change of muscle strength as a result of mandibular repositioning with a wax bite was reported by Smith.⁶⁴ He evaluated the muscle strength of professional football players by manual muscle testing and with the Cybex II dynamometer. The players were tested with their teeth together, with the teeth in a new postured wax bite position, and with a stock tray mouthguard. The mandible was positioned with the wax bite in its physiologic rest position toward the closest speaking space, and

with the midlines evenly aligned. Of the twenty-five players tested manually, ten were significantly stronger, twelve were slightly stronger, and three showed no change; none were weaker. With the stock tray mouthguard, three were significantly stronger, ten slightly stronger, nine showed no change, and three were weaker. Nine players were tested on the Cybex II comparing wax bite vs. teeth together. With the wax bite four tested stronger, two showed no change, and three were weaker. The four who tested stronger on the Cybex also tested stronger on the manual muscle test. Of the two showing no change, one tested slightly stronger on the manual muscle test. Of the three who tested weaker on the Cybex, two showed no change on manual muscle testing. In this limited sample, Smith gives a correlation of approximately 90% between those who tested stronger with manual muscle testing and the Cybex II dynamometer testing.

In our laboratory we have seen poor correlation between manual muscle testing and Cybex II dynamometer testing. (This is discussed in Chapter 15 of Volume I.) Since there were no controls run in Smith's study, it is questionable whether there was increased strength from the expectation of the player when wearing the wax bite. Additional information could have been derived if strength had been tested on the Cybex with the player using the stock tray mouthguard.

There is much disagreement and lack of general knowledge among physicians about the stomatognathic system. The introduction presented above from the literature certainly indicates there are remote health problems from temporomandibular joint dysfunction, and an influence on it from remote dysfunction in the body. Lack of consensus of opinion about the TMJ dysfunction syndrome is revealed in a survey³⁷ in which the professional respondents (M.D.'s and dentists) were not even in agreement that there is a TMJ dysfunction syndrome. The questionnaire asked if the respondent felt there is actually a distinct clinical syndrome comprised of (a) pain in the region of the TMJ, ear, face, neck and/or head; (b) noises associated with temporomandibular movement (cracking, popping); (c) subluxation/dislocation of the mandible; (d) limitation/deviation of mandibular movement; and (e) difficulty in mastication of food. Of the medical respondents, 51% believed there is, 6% that there is not, and 43% were uncertain. The dental group was composed of general practitioners and those in various specialties, including dental education. The approximate responses were: 76%, yes; 9%, no; and 14%, uncertain.

One of the major reasons for the controversy is that there are usually no organic findings in most

stomatognathic disturbance.^{9, 75} Most physicians are trained primarily to differentiate and treat pathological problems. Those who think in terms of functional disturbances are more likely to observe for and ultimately treat the functional type of disturbances found in the stomatognathic system.

Objective tests are needed to delineate the apparently widespread conditions that develop in the stomatognathic system, and also the remote conditions which can cause disturbance in the system. Bessette et al.⁹ designed an electrophysiological test to record the silent period of the masseter muscle following a jaw-jerk reflex. The latency was recorded for patients with TMJ syndrome and for normal subjects; it was considerably longer for the TMJ subjects. When compared by the student *t* test the latency was found to have a statistically significant difference at the 0.001 level. After treatment with a maxillary occlusal splint which was worn for three weeks, the TMJ patients' silent period returned to normal. Studies such as this, and the electromyographic study done at the Chicago College of Osteopathy⁷⁰ regarding leg length influence on the muscles of mastication, need to be expanded to put this primarily functional disturbance into perspective. Continued research of the mechanisms of manual muscle testing in evaluating the stomatognathic system provides an improved objective understanding of the disturbances in this area.

One of the problems of understanding the stomatognathic system is that a group of factors are being studied, usually as a single entity. Farrar and McCarty,^{22, 23} discussing the "TMJ Syndrome," point out that "The TMJ, like other joints and other parts of the human body, is subject to a variety of afflictions. (Who has ever heard of Hip syndrome? or the Knee syndrome?)" To this might be added that the stomatognathic system — in the broad sense — is subject to a great variety of afflictions. The structures within the system interact, and a disturbance in one area can, by a domino effect, create a great many additional disturbances. Often the secondary factors are treated, rather than the primary condition. Unfortunately, many who specialize in the treatment of TMJ disturbance have a concept that there is one primary cause of the condition and fit most patients into that slot. Because of the great amount of interaction within the system, the kind of condition the physician usually treats may be found, but it may not be the primary condition. It is important that a thorough examination protocol be developed, taking into consideration the wide range of etiologic factors in this condition.⁷⁵

The patient recognizing a symptomatic pattern often chooses his own diagnosis and treatment with

his selection of a physician. Treatment may include occlusal therapy, cervical or cranial manipulation, medication, psychological counseling, biofeedback, surgery, etc. One of these therapies may actually be appropriate for the patient, but it should be the job of the physician consulted as the primary care provider to diagnose the condition and determine the approach needed for its correction. Since no profession is capable of providing all the potential therapies which might be needed, consultation and referral are often the duty of the primary care physician. It is important to emphasize again that very often the symptomatic pattern presented by the patient is an effect rather than the primary cause; in fact, sometimes the patient's symptoms are iatrogenic from the treatment for some other condition.

The importance of studying the anatomy and physiology of the stomatognathic system to understand its interrelationship is pointed out by Dawson.¹⁴ Such study is essential because of the tremendous integration of the structures and function, both intrinsic and extrinsic to the system. He further shows how little it takes to throw the system out of balance. Referring to his specialty of occlusion, he states, "If there has been any single most important shortcoming in the field of occlusal problems, it has been the failure to understand how little it takes to throw the system out of balance."

There are several reasons applied kinesiology is an ideal tool for evaluating the stomatognathic system. (1) AK primarily evaluates function. The stomatognathic system first creates problems primarily by dysfunction — less often by pathology. (2) The stomatognathic system can disturb remote function, and AK has systems to evaluate function throughout the body. (3) Remote dysfunction can adversely influence the stomatognathic system and create secondary problems there. In most cases, AK can determine if the disturbance is primary or secondary.

The stomatognathic system can be evaluated very effectively with applied kinesiology methods. Correction of the system may be an absolute necessity in regaining optimum function throughout the body. There are many occasions where the primary disturbance is found in the stomatognathic system; until it is corrected, other areas either will not hold their corrections or cannot be corrected. The same basic advantages found in the use of applied kinesiology in other areas of the body are present in its application to the stomatognathic system; it aids in (1) finding areas that are dysfunctioning, (2) determining the necessary correction, (3) evaluating whether the correction was effective, and (4) determining whether the correction is of a

Stomatognathic Area Introduction

lasting nature. This breaks down the TMJ syndrome, dental distress syndrome, myofascial pain-dysfunction, or whatever it is called, into a specific diagnosis

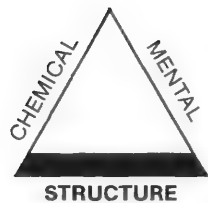
to break the vicious circle so often present, thus avoiding the treatment of symptoms and secondary conditions.

Triad of Health

The cause of dysfunction in the stomatognathic system can be found in any one or more sides of the triad of health. Most of this discussion thus far has

related to structural factors. A brief expansion of possible initiating disturbances will help put this into perspective.

STRUCTURE



One of the most common approaches to disturbance in the stomatognathic system is balancing the dental occlusion. A brief explanation of the physiology of malocclusion will help explain how equilibration of the occlusion can improve function in remote areas. Malocclusion stimulates the proprioceptors in the periodontal ligament which, through integrating neuronal pathways, pattern the muscles of mastication to develop a new closing pattern. This guides the teeth into proper occlusion, thus eliminating improper tooth contact. The result may be tension on some of the muscles of mastication which disturbs the closed kinematic chain of the stomatognathic system. What may have been a small premature tooth contact has now caused a muscular imbalance which might cause a change in stimulation to the equilibrium proprioceptors residing in the muscles balancing the head on the neck and the neck on the body. Obviously, disturbances will develop in the neurologic mechanisms which orient the body in space. Orthopedic problems may result, but possibly more significant are spinal subluxations which disturb nerve function and cause health problems almost anywhere in the body.

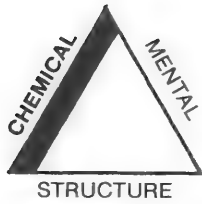
Another possible result of malocclusion is that every time an individual chews, the very powerful muscles of mastication pull on their cranial origins in an imbalanced manner, creating or perpetuating cranial faults. This, again, can cause disturbance in the nervous system, with a wide range of influence.

Structural problems, on the other hand, can

develop in the feet, pelvis, or other structure(s) below the stomatognathic system. Proprioceptive communication from the feet provides information through the neuronal pools to the sternocleidomastoid, upper trapezius, and other gait muscles which can disturb balance in the closed kinematic chain of the stomatognathic system, ultimately influencing the muscles of mastication and causing malocclusion. We see the malocclusion as a secondary factor. If equilibration is done with the muscular imbalance present, it may relieve some symptoms in the TMJ or other areas of the stomatognathic system, but the basic underlying cause of the problem remains. If the body — a self-correcting, self-maintaining mechanism — is capable of correcting the foot, pelvis, or whatever was primary, there will now be an iatrogenically produced malocclusion because it was not recognized as a secondary factor. If the body cannot correct the primary area, it is quite likely that another physician will correct the patient's complaint of low back or foot pain and thus correct the primary area of involvement. Again a malocclusion results because the equilibration was done in the presence of imbalanced muscles. It is now possible that symptoms in the TMJ or other area of the stomatognathic system may reappear, or some may occur which were not present in the first place.

Each of these factors, and more on structural integration, will be discussed regarding examination and correction later in this section and in Section III. It is important now to briefly put the other two factors of the triad of health into perspective with the stomatognathic system.

CHEMICAL



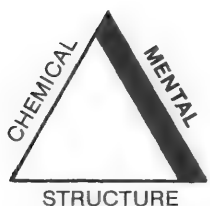
The stomatognathic system can be thrown out of balance, either directly or indirectly, by the chemical side of the triad of health. Nutritional imbalance or harmful chemicals can disturb muscle balance as well as the cranial-sacral primary respiratory mechanism. It is not clear how this disturbance develops but, on a clinical basis, it is often seen that antacids cause imbalance in the cranial primary respiratory mechanism. Cranial faults will quite often disturb the occlusion in TMJ function; this will be more thoroughly discussed later.

It has been clinically observed in applied kinesiology that a sacroiliac dysfunction is frequently associated with a temporomandibular joint disturbance.³³ The type of involvement is usually a category II pelvic subluxation as a result of a weak sartorius and/or gracilis, which appear to relate with adrenal function. Often it is clinically seen that sartorius and gracilis muscle function improves with nutrition designed to aid adrenal function. These muscles are also weakened sometimes as a result of chemicals or food substances which are detrimental to adrenal function. This muscle weakness and the subsequent category II pelvic subluxation represent remote structural imbalance causing stomatognathic

system involvement on a secondary basis. This may relate with a study done by Evaskus and Laskin¹⁸ of thirty-two patients with myofascial pain-dysfunction syndrome (MPD). They evaluated the urinary concentrations of catecholamines and 17-hydroxy steroid levels. Although this study was done to evaluate stress levels in patients with MPD syndrome, it appears to have value in relating the clinical adrenal dysfunction, noted above, with disturbance in the stomatognathic system. Further evaluation relating sartorius and gracilis dysfunction with category II and levels of 17-hydroxy steroids and catecholamine levels is indicated to determine if there is a statistically significant correlation.

There is evidence that certain types of nutritional deficiencies can contribute to periodontal disease, with resulting unstable teeth which disturb the occlusion.¹¹ One of the most important dietary factors of periodontal disease appears to be a blood sugar handling stress, with a tendency toward a low blood sugar level. There is a correlation, especially in applied kinesiology, of functional adrenal insufficiency with chronic blood sugar handling stress. This may indicate a correlation between the alarm state of adrenal function noted by Evaskus and Laskin¹⁸ with MPD, and the often observed sartorius, gracilis, and category II disturbances observed in applied kinesiology, with stomatognathic disturbance.

MENTAL



Much disturbance in the stomatognathic system has been blamed on anxiety; in fact, there is considerable controversy within the dental profession as to whether malocclusion or an emotional problem is the primary cause of temporomandibular joint disturbance.

Anxiety, tension, and stress and their influence on temporomandibular joint pain and dysfunction were studied by Solberg et al.,⁶⁸ using the Minnesota Multiphasic Personality Inventory. They compared a control group with a symptomatic group which displayed (1) pain in the TMJ, muscles of mastication, or associated structure for which no observable cause could be found, and/or (2) limitation of normal range of mobility of the mandible. There was greater anxiety in approximately half of the symptomatic group than in the control group; however, the elevated anxiety in the majority was within normal limits.

Stress that produced stomatognathic disturbance

and stress caused by stomatognathic disturbance have been reported by many.^{26, 43, 55} Although it is obvious that in some cases the mental side of the triad of health is a primary etiology, it is more likely a combination of factors. Weinberg⁷⁵ emphasizes that it is meaningless to attempt to categorize temporomandibular joint dysfunction patients as suffering either from emotional stress or from occlusal disharmony; the two factors are intimately related and cannot be viewed in isolation.

In applied kinesiology it is observed that dysfunction of the stomatognathic system is often the cause of neurologic disorganization. As discussed in Volume V of this series, many individuals who have emotional disturbances do so because of some functional disturbance in the body. One very common cause of emotional problems is neurologic disorganization. It is clinically observed that when the functional disturbance is corrected, the same stresses that previously bothered the individual are no longer such a problem. The person is now able to cope with the

Stomatognathic Area Introduction

stresses of life that were previously unbearable. When uncorrected, a vicious circle may develop. When there is either stress, tension, or emotional problems, there is often bracing of the jaw.^{59, 78} If there is an imbalance of muscular contraction, or if there is malocclusion, the stresses put into the cranium can create or perpetuate cranial faults, causing further neurologic disorganization which may have been contributing to the inability to cope in the first place.

It must be recognized that any side of the triad of health can be the initiating factor of disturbance in the stomatognathic system. Laskin⁴³ presents strong evidence for a psychophysiological theory of myofascial pain and dysfunction. He cites studies done at the Temporomandibular Joint Research Center, University of Illinois, which included epidemiologic,⁴⁴ radiologic, biochemical,^{5, 18} and physiologic studies^{36, 45, 46, 71} which indicate that the mental aspect is primarily responsible for the MPD syndrome.

It is difficult to place into perspective the primary cause of dysfunction in the stomatognathic system because of the tremendous interaction among its parts. There is no question that stress is very common in patients with this type of disturbance.

Farrar²³ points out that "... the relationship between stress and TMJ disease has not been brought into sharp focus, primarily because TMJ patients have been lumped into one major category without regard to differentiation and also because most researchers in this field have assumed that stress causes TMJ problems. They have not attempted to research the idea that the temporomandibular joint problem causes stress reaction."

The question of which side of the triad of health is primarily responsible for the dysfunction seems to be better understood with applied kinesiology examination, since it deals with the interaction of all three sides. Clinical evidence appears to indicate that no particular side of the triad is always primarily responsible. Most chronic patients show involvement with two or all three sides of the triangle. Treatment directed to one side may provide a certain amount of relief, even though the primary involvement is not treated. More study is required to determine the role played by stress in disturbance of the stomatognathic system. Using relaxation techniques, Gessel and Alderman³¹ had good results with patients who did not have depressive symptomatology. There were poor results with those who were depressed.

Summary

This brief introduction attempts to put into perspective the wide range of interrelationships within the stomatognathic system, and its interrelationship with the rest of the body. There are many diverse opinions regarding this mechanism, the etiology of dysfunction, and what influence it has on body dysfunction. It seems apparent that these diverse opinions arise from failure to definitively diagnose the basic underlying cause and effectively treat it to eliminate the effects. As long as effects and

symptoms are being treated, the primary condition is perpetuated and probably intensified. The applied kinesiology approach to evaluating this system and its many interrelationships helps put this into perspective for quick, efficient diagnosis, correction, and re-evaluation. This approach should be used by an individual thoroughly knowledgeable about the anatomy and physiology of the stomatognathic system and the rest of the body, and in applied kinesiology procedures.

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Stomatognathic Area Introduction

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Chapter 9

Anatomy of Muscles of Mastication, Tongue, and Teeth

Introduction

It is important that the applied kinesiologist have an excellent working knowledge of all the muscles in the stomatognathic system. The primary muscles of posture, which include the cervical flexors and extensors, are presented in Volume I. This chapter covers the muscles of mastication and the tongue; the hyoid muscles are discussed in Chapter 13.

Understanding the anatomical attachments and the course of a muscle is the first step in developing a working knowledge of these structures. It then becomes necessary to learn the firing pattern of the muscle as indicated by electromyographic study. When individual muscles are understood, a study of combined muscle action will reveal the function in this area. Unfortunately, most anatomy texts deal with muscles individually; because the action is given for each one, there is often a failure to recognize the complex synergism present in this area. In no action of temporomandibular joint function is only one muscle responsible for the activity; in fact, it becomes difficult to determine which muscle is most important in a specific activity. One particular muscle may produce the greatest amount of power, but if it does not have the synergistic activity of a lesser muscle, no activity may take place at all; if it does, it may cause severe stress in the mechanism. The stomatognathic system has a prodigious nerve supply; repetitive failure of one muscle to respond appropriately can cause devastating results almost anywhere in the body.

The complexity of muscular activity in the stomatognathic system can be likened somewhat to the synergistic activity necessary in shoulder movement. The muscles listed as prime movers in shoulder abduction are the supraspinatus and deltoid,

but without activity from the small and seemingly insignificant subclavius, full shoulder abduction cannot take place. The activity is also dependent on the rotator cuff muscles, divisions of the trapezius, levator scapula, serratus anticus, the rhomboids, and the pectoral group. What may seem the simple activity of abducting the shoulder becomes very complex. So it is with the temporomandibular joint and the activity of mastication. It becomes even more important to understand the firing activity and synergism of the muscles of mastication because there is limited ability to test these muscles by manual muscle testing; this is easy with the muscles for shoulder abduction. Indirect methods of testing the masticatory muscles require that a physician understand which muscles are synergistic in normal activity, and also how the muscles can change their firing activity in abnormal function.

So far this discussion has been limited to the muscles of mastication. Their activity must be integrated with the activity of the hyoid muscles and cervical flexor and extensor muscles, because the muscles of mastication are only a part of a closed kinematic chain. If normal function is lost in any aspect of this chain, disturbance will probably result in other muscle dysfunction, contributing to make a complex problem.

The general anatomy of the tooth and periodontal ligament is also discussed in this chapter. The proprioceptors in these ligaments supply afferent impulses, influencing the way in which the muscles of mastication work in a synergistic manner to control mandibular movement. This neurologic mechanism is considered the neuroanatomic basis of mandibular movement.

Format of Masticatory Muscles

First the anatomy^{10, 17, 28, 41, 68, 74, 79} will be described, giving the origin, insertion, and general description of the muscle. In the applied kinesiology examination and treatment of these muscles, it is necessary to locate various areas of the muscles by palpation. Often the structure is difficult to contact, so an excellent knowledge of the muscle is necessary. The general action of the muscle as described in anatomy texts is given, correlated with the physiology of activity as observed on electromyographic

studies. Kinesiology of the masticatory muscles is the same for children and adults.⁷²

The neurolymphatic^{22, 50, 73} and neurovascular^{4, 5, 23, 73} reflexes and stress receptors,^{24, 73} as well as the nerve supply, are the same for all muscles and will be given with each muscle. Under "General Discussion," body language of involvement and other information of general interest regarding the muscle will be presented.

TEMPORALIS

Origin: From the whole of the temporal fossa, excluding the part formed by the zygomatic bone, and the deep surface of the temporal fascia.

Insertion: By a tendon to the medial surface, apex, anterior and posterior borders of the coronoid process, and the anterior border of the ramus of the mandible, nearly as far as the last molar tooth.

Description: The temporalis is a broad, fan-like muscle. The fibers converge to form a tendon which passes deeply to the zygomatic arch to its insertion at the coronoid process. The temporal fascia covers the temporalis and is a strong, fibrous sheet aponeurotic in appearance. Because of its thickness, it is difficult to palpate the relaxed temporalis muscle; when there is hypertonicity or contraction the borders are easily felt.

The temporalis is typically divided into three sections — the anterior, middle, and posterior fibers. The anterior fibers are directed superiorly and somewhat anteriorly. The medial fibers are then directed mostly superiorly and posteriorly, while the posterior fibers are directed mostly posteriorly and somewhat superiorly. The posterior fibers are almost parallel to the occlusal plane.⁶³

The most posterior fibers of the temporalis, running primarily horizontally, bend sharply inferiorly at the root of the zygoma to insert on the mandible.¹⁶ These fibers appear to be important in stabilizing the condyle and disc in the mandibular fossa to help maintain physiologic centric relation (see page 327).

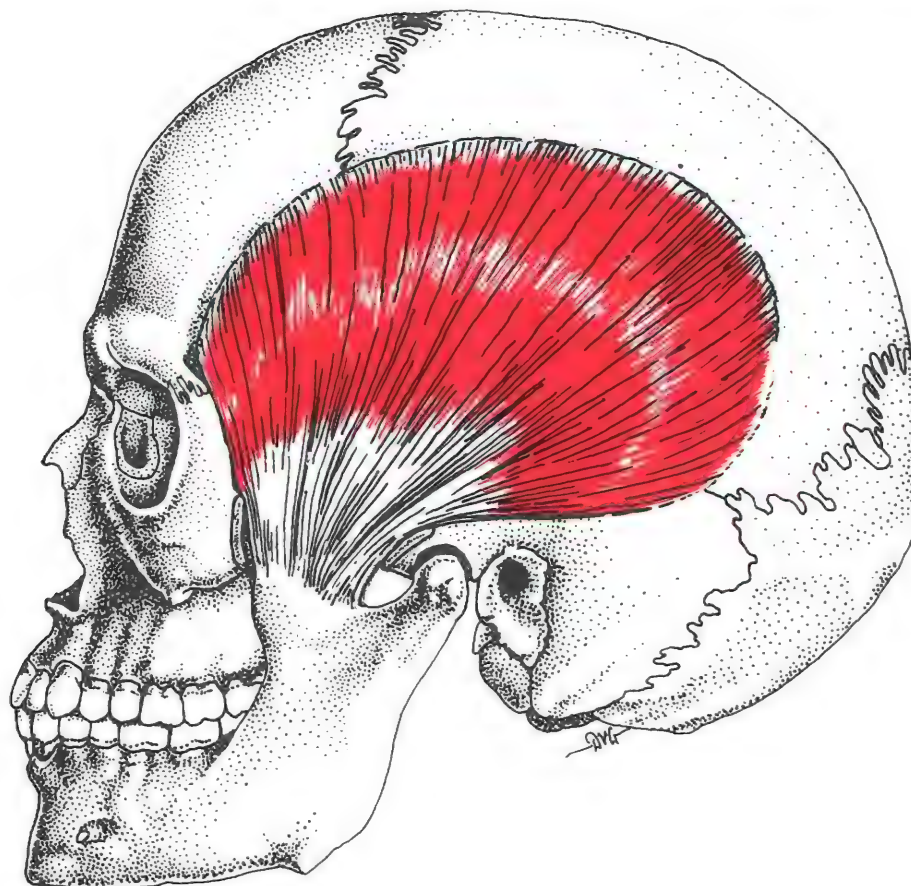
Action: Elevates the mandible to close the jaws. The posterior fibers retract the mandible. The fact that the three divisions of the temporalis muscle act

separately may be explained by their usual nerve supply — three divisions of the deep temporal branch of the anterior trunk of the mandibular nerve.^{43, 44} The resting "tonus" of the temporal muscle occurs in all three parts. It seems there is stronger resting activity in the posterior fibers^{37, 44} which may relate to the inferior angling of some of the posterior fibers in stabilizing the condyle and disc into the articular fossa. The amount of activity of the temporalis in the rest position is important in applied kinesiology because the temporal tap⁷³ and TMJ function appear to be influenced by hypertonic posterior temporalis fibers.

In ordinary jaw opening there is no activity in the temporalis muscle.³⁷ Although this seems obvious, there are some activities of the masticatory muscles that one would not expect from their anatomical arrangement.

In maximum mouth opening there is activity in the anterior fibers and a greater amount in the posterior fibers.³⁷ This is apparently an activity to protect the temporomandibular joint; it may be important in some cases of muscle reactivity.

Lateral movements of the mandible are described by various terms, depending on an author's area of interest. For example, anatomists refer to lateral movement as abduction of the menton and prosthodontists to lateral excursive movement. When referring to the grinding movements of chewing (a lateral movement), trituration is often used. Here abduction refers to the lateral mandibular movement. Electromyography generally shows the temporalis to be active in mandibular abduction in a normal individual. The posterior fibers of the temporalis are active on the side of mandibular abduction.^{37, 44, 71} When the mandible returns from lateral abduction, the anterior



9—1. *Temporalis muscle.*

and posterior fibers on the opposite side are active.³⁷ In an activity where the mandible moves from side to side, first abducting to one side and then to the other such as is used in applied kinesiology examination, both temporalis muscles are activated. The temporalis contribution to the movement is primarily from the posterior fibers on both sides.

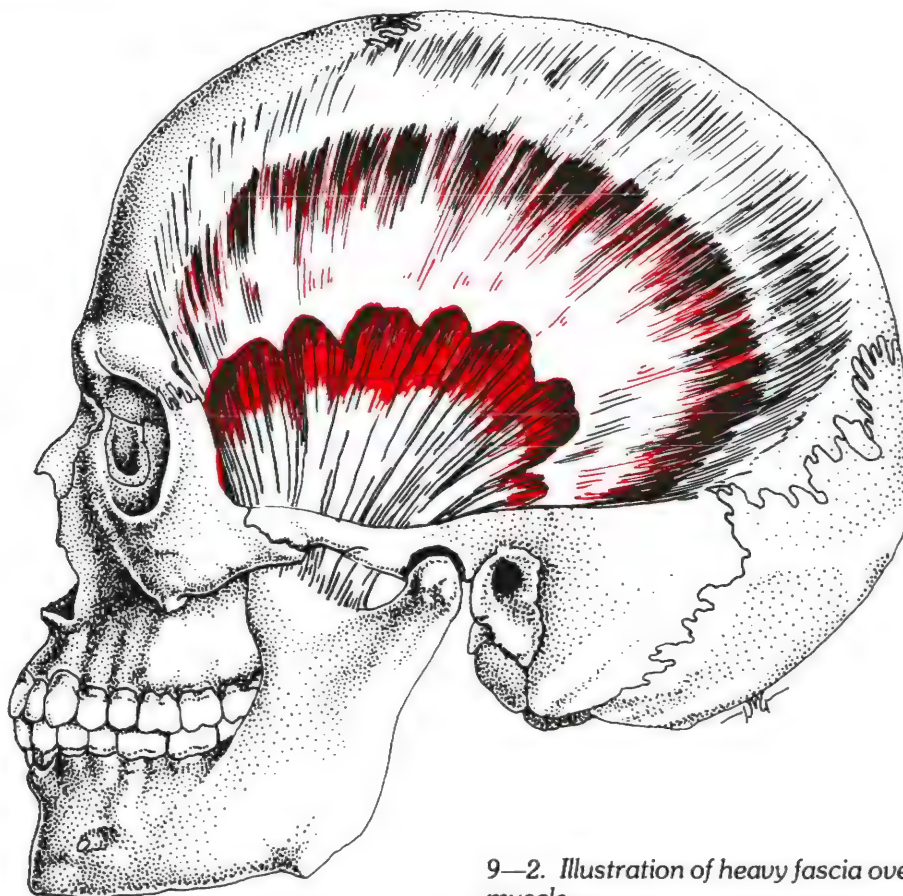
The anterior and posterior bellies of the temporalis muscle have reciprocal inhibition³⁶ because of the antagonistic activity of the anterior and posterior bellies. This may be important on a reactive muscle basis during applied kinesiology examination. It is especially applicable when there is disturbance from moving the mandible from side to side, such as in “wagging” the jaw.

The temporalis contributes differently in active biting, depending on whether the contact is with the molars or with the incisors. During an end-to-end incisor bite all fibers are active, with more activity in the anterior division. The major activity of the incisor

bite is contributed by the masseter,^{37, 39} which contracts first.⁶⁹ It is likely that the contribution of the temporalis is primarily that of guiding and regulating the bite. In forced molar occlusion, the activity is over all parts of both the masseter and the temporalis muscles.³⁹

There is general agreement that the anterior fibers are not active in straight protraction of the jaw.³⁹ This has been the subject of some controversy among electromyographers.

The activity of the temporalis in retraction of the mandible has become important in certain conditions observed in applied kinesiology. There is no argument about this strong activity in electromyographic studies.^{2, 39, 45} It appears to be present regardless of malocclusion or other functional changes that might influence muscle activity. It is observed that retraction from the resting position produces little movement of the mandible, as it can ordinarily be displaced very little.⁴⁴ If retraction from this position



9—2. Illustration of heavy fascia over the temporalis muscle.

is attempted, the posterior fibers are primarily active. If retraction is done from the fully protruded position, all fibers of the temporalis are active.

Neurolymphatic:

Anterior: 2nd, 3rd, and 4th intercostal spaces adjacent to the sternum.

Posterior: At T2, 3, and 4 near the laminae.

Neurovascular: Ramus of the mandible below the zygoma.

Stress Receptor: In transverse plane, approximately 1" above glabella.

Meridian: Stomach.

Nerve Supply: Deep temporal branches of the anterior trunk of the mandibular nerve.

General Discussion: The most frequently involved portion of the temporalis muscle on an applied kinesiology basis is the posterior division. Often there is a need for "setting down" the neuromuscular spindle cell. When there is dysfunction of a neuro-

muscular spindle cell, its location can usually be readily found by palpation. The area feels nodular, ropy, or somewhat congested. It will show positive therapy localization and will usually be tender.⁷³

The temporalis muscle and its fascia are often the site of trigger points which may need spray and stretch technique.⁷³ For optimum stretch of the posterior fibers, the jaw should be opened with protrusion. This may require the physician to contact the anterior lower dental arch and mandible, applying gentle traction with opening of the mouth.

Fascial flush technique⁷³ is occasionally needed on this muscle. The clinical indication is muscle stretch reaction; the fascia palpates as tense, with minimal mobility.

With hypertonicity of the temporalis muscle there is often a jamming of the squamosal suture; it may need rather forceful separation. Examination for squamosal suture jamming should be done after the temporalis muscle has been relaxed and, as usual, should be done by challenge.

Recurrent hypertonicity of the temporalis is often secondary to emotional "bracing." The emotional stress factor can be primary or secondary; thorough examination to reveal its status is necessary.

Anatomy of Muscles of Mastication, Tongue, and Teeth

MASSETER

Origin:

Superficial layer: Thick aponeurosis from the zygomatic process of the maxilla; anterior two-thirds of the lower border of the zygomatic arch.

Middle layer: Deep surface of the anterior two-thirds of the zygomatic arch and from the lower border of the posterior one-third.

Deep layer: Deep surface of the zygomatic arch.

Insertion:

Superficial layer: Angle and lower half of the lateral surface of the ramus of the mandible.

Middle layer: Middle of the ramus of the mandible.

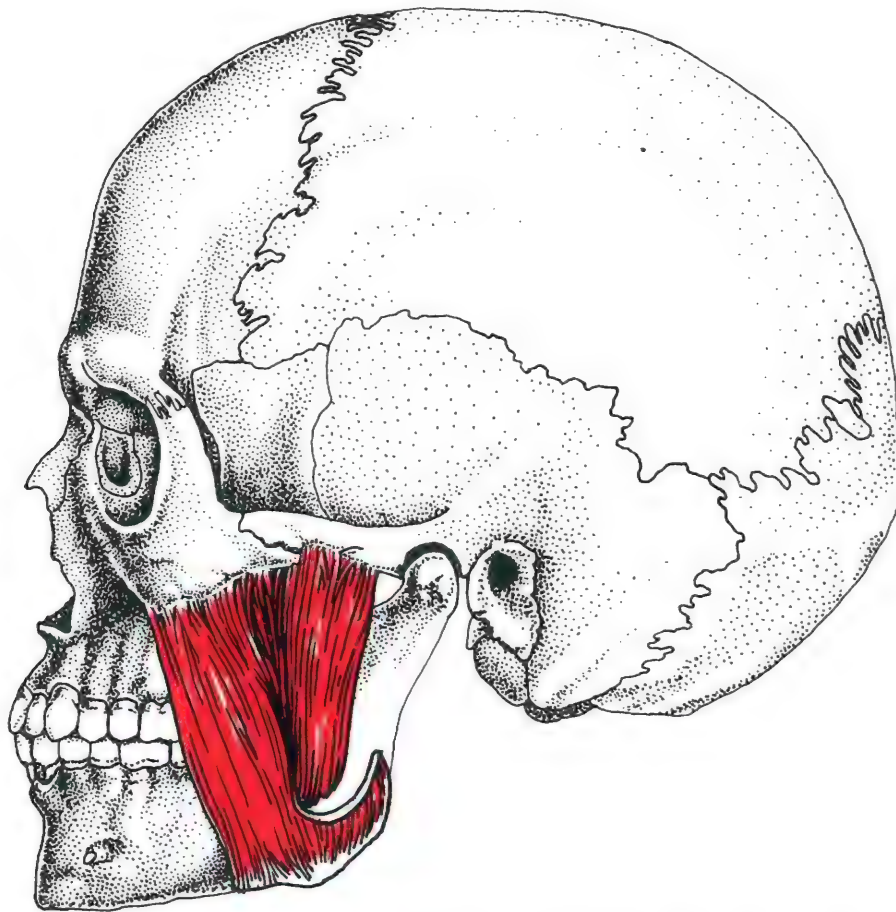
Deep layer: Upper part of the ramus of the mandible and into the coronoid process.

Description: The overall masseter muscle is basically rectangular. The deep and middle layers make up the deep part of the masseter. The larger superficial

portion primarily covers the deep portion; it is fused anteriorly with it, but separated posteriorly. The two layers making up the deep portion form a cruciate muscle, which means the fibers cross each other similar to the letter "x." Overall the muscle is formed by an intricate arrangement of tendinous and fleshy bundles. The net effect of this construction is to make the muscle extremely powerful.⁶⁸

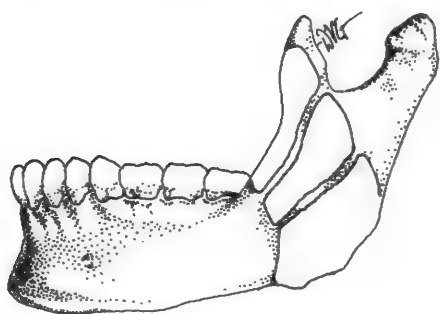
Action: The general action of the masseter muscle is to elevate the jaw and clench the teeth.

The masseter muscle has minimal or no activity in the resting position.² It has maximum activity during incisor biting.³⁹ There is some activity during protrusion, and the deep fibers are occasionally active on retraction.^{9,39} There is no activity observed on abduction.⁵⁶ Other than its major activity in closing the jaws and giving force to occlusion and mastication, the masseter is not greatly active. Probably its limited activity in other motions is for



9—3. Masseter muscle with superficial layer sectioned to show middle and deep layers.

Anatomy of Muscles of Mastication, Tongue, and Teeth



9—4. Three insertions of masseter muscle.

guiding and stabilizing the mandible's activity.

In a study of the action of the temporalis and masseter muscles during the chewing of hard and soft foods,⁶⁹ it was found that the masseter always started the mandibular elevation, and was followed by the temporalis. When chewing carrots, the delay of the temporalis was shorter than when chewing soft white bread. The chewing activity of individuals varied, but there was an habitual pattern characteristic to each individual. It seems that the major power of mastication was from the masseter; the following action of the temporalis was for stabilization of this action by first the anterior and later the posterior bellies. It appears that this individual action of temporalis stabilization is an adaptation for the characteristics of occlusion in the particular individual.

Neurolymphatic:

Anterior: 2nd, 3rd, and 4th intercostal spaces adjacent to the sternum.

Posterior: T2, 3, and 4 near the laminae.

Neurovascular: Ramus of the mandible below the zygoma.

Stress Receptor: In transverse plane, approximately 1" above glabella.

Meridian: Stomach.

Nerve Supply: Masseteric nerve from the anterior trunk of the mandibular division of the trigeminal nerve.

General Discussion: The masseter muscle appears to often have disturbance in its neuromuscular spindle cells. This is probably because of the great power this muscle can develop in normal actions and in bracing and bruxism, which may either be clenching during emotional stress or nocturnal tooth grinding.^{65, 66, 80}

The apparently dysfunctioning neuromuscular spindle cell in the masseter can be readily located by palpation. This is one of the easiest areas in the body on which to make this type of examination. The muscle may be exquisitely tender in this area; therapeutic efforts should be initiated with caution because of pain to the patient. In this case, spray and stretch technique is often effective in reducing the pain; however, it may not correct the disturbance. After reducing the pain, it is often possible to apply the usual treatment for neuromuscular spindle cell dysfunction.

As with the temporalis, if there is evidence of emotional bracing, investigation should be done to find the basic underlying cause. Dysfunction of the masseter which is secondary to bracing will respond only temporarily to therapeutic efforts directed to the muscle (for further information on bruxism and bracing, see page 374).

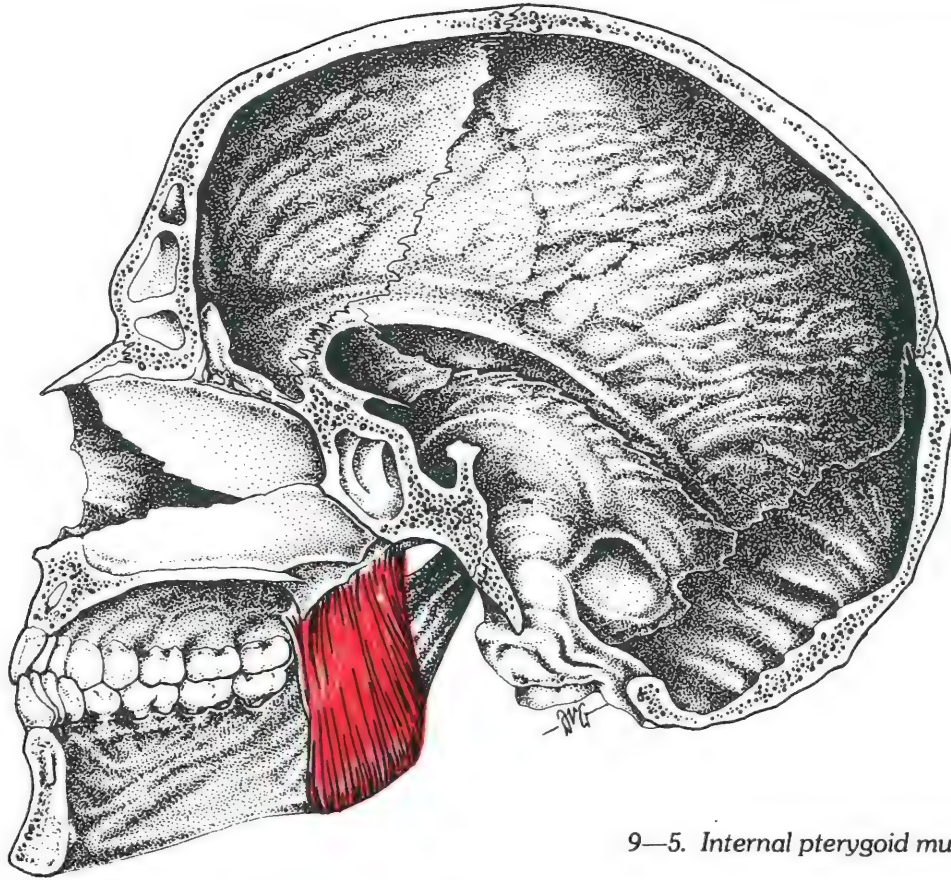
The masseter has a major influence on the cranial primary respiratory system. Its origin from the zygomatic arch is a great lever into the cranial closed kinematic chain. In full occlusion, the origin and insertion are reversed. The temporal and zygomatic bones become the structures which potentially can be moved in a micro fashion, activating the cranial primary respiratory mechanism in either a favorable or an unfavorable manner. If the force directed into the cranium is balanced, there will be favorable activation; on the other hand, if there is malocclusion or muscle dysfunction causing the force to be imbalanced, cranial faults can either be created or perpetuated by activity of the masseter muscle.

INTERNAL PTERYGOID (Medial Pterygoid)

Origin: Medial surface of the lateral pterygoid plate and the pyramidal process of the palatine bone. A more superficial slip from the lateral surfaces of the pyramidal process of the palatine bone and tuberosity of the maxilla.

Insertion: Inferior and posterior parts of the medial surface of the ramus and the angle of the mandible.

Description: A rectangular muscle which is the counterpart of the masseter. Its fibers pass from the



9—5. Internal pterygoid muscle.

origin infralaterally and posteriorly. Like the masseter, its construction is characterized by alternation of fleshy and tendinous parts.⁶⁸

Action: Protracts and elevates the mandible and is active in rotary motion while chewing. The activity during protraction is less if there is mandibular depression prior to the protraction.⁴⁴ In abduction of the mandible, there is activity of the contralateral muscle.^{9, 44}

Neurolymphatic:

Anterior: 2nd, 3rd, and 4th intercostal spaces adjacent to the sternum.

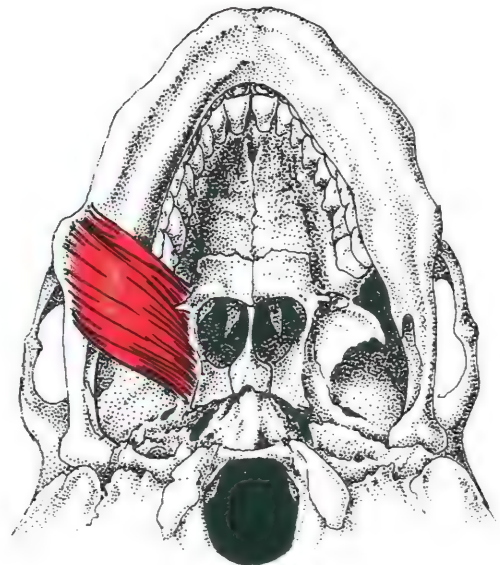
Posterior: Between T2, 3, and 4 near the laminae.

Neurovascular: Ramus of the mandible below the zygoma.

Stress Receptor: In transverse plane, approximately 1" above glabella.

Meridian: Stomach.

Nerve Supply: Medial pterygoid nerve of the mandibular division of the trigeminal nerve.



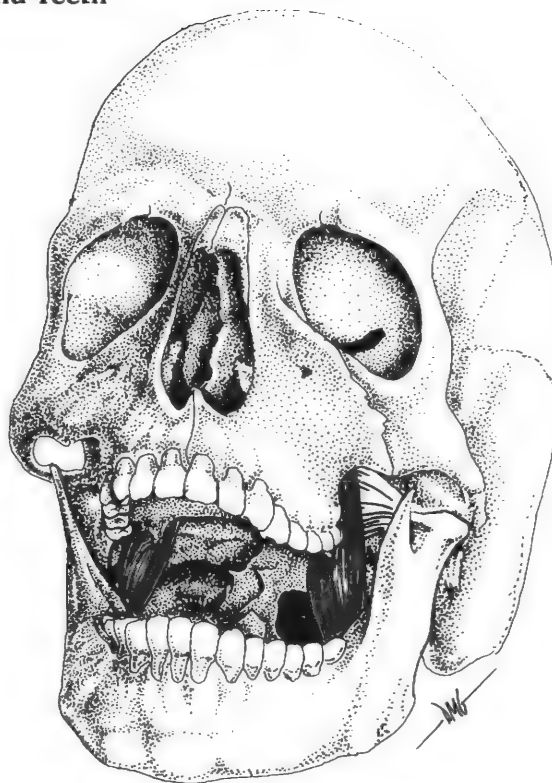
9—6. Internal pterygoid muscle.

Anatomy of Muscles of Mastication, Tongue, and Teeth

General Discussion: The internal pterygoid is very active in the process of mastication and, although not as often involved as the temporalis and masseter, quite frequently there is apparent dysfunction of the neuromuscular spindle cell. When this muscle is out of balance, the rhythm of the masticatory muscles is disturbed, causing occlusal problems. Its origin from the lateral pterygoid plate and pyramidal process provides this muscle with a lever entrance into the cranial closed kinematic chain. Imbalanced pull for whatever reason directly influences the sphenoid, a key bone in the chain which articulates with twelve other bones.

It is important for the physician to be familiar with the anatomy of this muscle so that it may be palpated for tenderness and contacted for treatment procedures.

To palpate the internal pterygoid,^{12, 13} have the patient's mouth open as wide as possible. Contact the muscle's insertion as inferiorly on the ramus of the mandible as possible. Progress up the muscle to the level of the pterygoid plate and move the tip of the finger medially across the anterior border of the internal pterygoid muscle. The muscle often feels very tense and ropy, and may be very tender.



9—7. Internal pterygoid muscle



9—8. Palpation of internal pterygoid. See also illustration 11—2.

MANDIBULAR SLING

The masseter and internal pterygoid muscles are so arranged that they suspend the angle of the mandible in a sling. This arrangement, with the highly mobile temporomandibular joint acting as a guide, enables great excursive movements in all directions

except posterior. The arrangement and interaction of these muscles, along with others, are programmed for the various activities required of this versatile structure.

Anatomy of Muscles of Mastication, Tongue, and Teeth

BUCCINATOR

Origin: There are three areas of origin — from the maxilla, pterygomandibular raphe, and the mandible.

Maxilla: From the buccal surface of the alveolar processes of the three molar teeth.

Pterygomandibular raphe: This is a tendinous inscription between the buccinator and constrictor pharyngis superior muscles which gives origin to the middle portion of the muscle. It is attached superiorly to the pterygoid hamulus and inferiorly to the posterior end of the mylohyoid line of the mandible.

Mandibular: The lateral alveolar processes inferior to the three molar teeth.

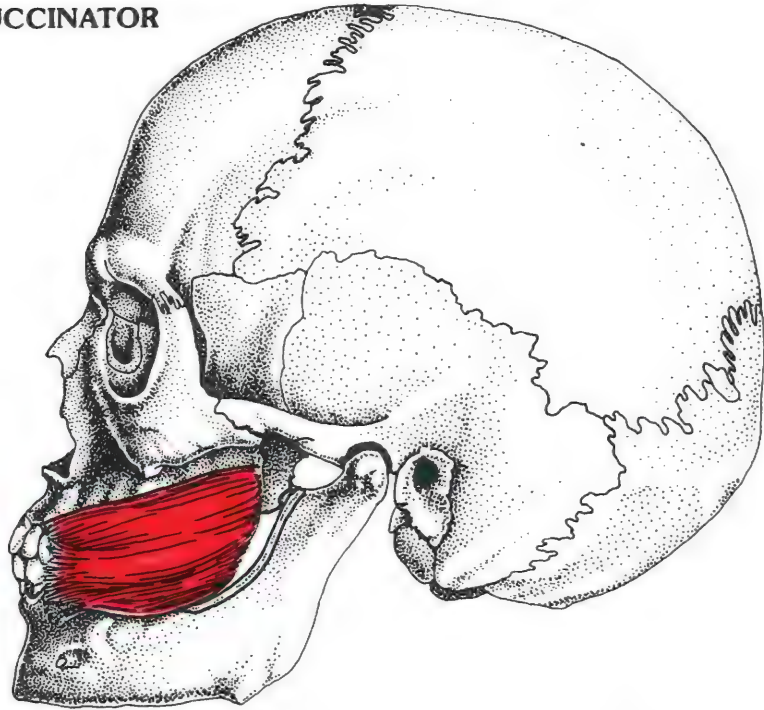
Insertion: Blends into the deeper layer of muscle fibers of the lips; splits in the middle to join with the orbicularis oris of the upper and lower lips.

Action: During mastication the buccinator acts to hold food between the teeth. Activity of the buccinator is necessary in combination with the tongue acting as a plunger for suckling. It also acts with the orbicularis oris to primarily form the outer envelope of the teeth; the inner envelope is formed by the tongue. The buccinator is the foundation from which the orbicularis oris functions. It compresses the cheeks to forcefully blow air, such as in trumpet playing.

Nerve Supply: Buccal branches of the facial nerve.

Test: Observe the patient's ability to contract the buccinator, placing external pressure against the lateral dental arches. Palpate the muscle for contraction. Another evaluation is to have the patient purse his lips and apply maximum air pressure. Palpate the buccinator bilaterally for contraction. An even better method is for the examiner to place his index finger between the teeth and cheek, and have the patient muscularly contract against the examining finger. Palpate for contraction of the buccinator.

Discussion: The buccinator appears to be involved on a reactive muscle basis with the masseter muscle in some cases of temporomandibular joint dysfunction. This same reactive muscle basis may be present



9—9. Buccinator muscle inserts into the lips primarily to the orbicularis oris.

with some of the smaller muscles of the lips, such as the depressor anguli oris, mentalis, zygomaticus major and minor, orbicularis oris, etc. Blanton and co-workers⁸ studied the buccinator with electromyography and found no activity during slowly opening and closing the mouth. During chewing patterns there was marked to very marked activity which was asynchronous with the activity of the masseter and temporalis muscles. There was negligible or moderate activity synchronous with the firing of the masseter and temporalis muscles. This is important in applied kinesiology evaluation because of the suspected reactive muscle relationship between the buccinator and the masseter. Unless the jaw moves rapidly or the patient chews during applied kinesiology evaluation, a reactive involvement may be missed. Of course, if the patient is chewing, a neurologic tooth or malocclusion must be ruled out of the condition.

The buccinator is a very important muscle in helping make up the outer muscular envelope surrounding the dental arches. Balanced strength and activity of the inner and outer envelopes are necessary for normal position of the teeth. The buccinator is especially important in the outer envelope because it is an important base from which the orbicularis oris functions. Without normal strength in the buccinator, the orbicularis oris will appear weak, even though it may not be (see Chapter 15 on "Myofunctional Therapy").

Anatomy of Muscles of Mastication, Tongue, and Teeth

EXTERNAL PTERYGOID (Lateral Pterygoid)

Origin:

Superior head: Infratemporal surface and infratemporal crest of the greater wing of the sphenoid.

Inferior head: Lateral surface of the lateral pterygoid plate.

Insertion: Anterior part of the neck of the condyle of the mandible and into the articular capsule and disc of the temporomandibular articulation.

Description: The muscle is short and thick with two parts or heads. The fibers pass posteriorly and laterally to the TMJ articulation. Some consider that this is, in reality, two muscles both functionally and structurally.²⁷

Action: Protrudes mandible, pulls articular disc forward, assists in rotary motion of chewing.

The external pterygoid is basically the jaw-opening muscle. During mandibular depression it provides the first muscular activity, and it reaches its peak before other jaw-opening muscles become active. Its activity then persists through the full jaw-opening motion.⁴⁴ The muscle's principal activity appears to be drawing the meniscus and condyle head anteriorly.

In man the external pterygoid muscle is difficult to study. In an effort to determine the activity of the two divisions, McNamara⁴² studied the muscle in the Rhesus monkey, which has an anatomical arrangement similar to man. He found that the inferior head (the larger head) acted in opening movements of the mandible; no activity was noted in closing movements or in swallowing. On the other hand, the superior head was not active during opening movements but was during closing movements of chewing and clenching the teeth, and during deglutition. He theorized the superior head positioned or stabilized the condylar head and disc against the articular eminence during closing movements of the mandible, while the inferior head assisted in the translation of the condylar head downward, anteriorly and contralaterally during opening movements. This is supported by Grant,²⁷ who studied the movement arms of both muscle heads which are collectively called the external pterygoid in man and in the Rhesus monkey. Particularly from the rest position, the superior head has the effect of closing the jaws while the inferior head has the effect of opening them. The two heads are antagonists; thus the external pterygoid must be considered as two muscles.

In straight protraction, both lateral pterygoid muscles are active along with the masseter and

internal pterygoid.^{42, 44} In abduction the contralateral inferior external pterygoid is active;⁴⁴ thus in repeated right and left abduction, the inferior external pterygoids are alternately active.

Neurolymphatic:

Anterior: 2nd, 3rd, and 4th intercostal spaces adjacent to the sternum.

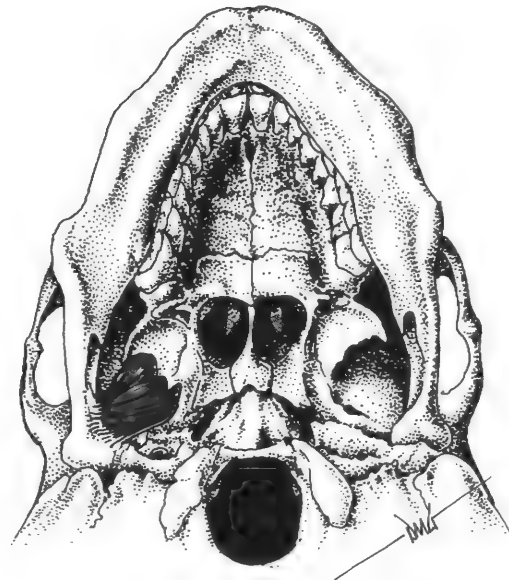
Posterior: Between T2, 3, and 4 near the laminae.

Neurovascular: Ramus of the mandible below the zygoma.

Stress Receptor: In transverse plane, approximately 1" above glabella.

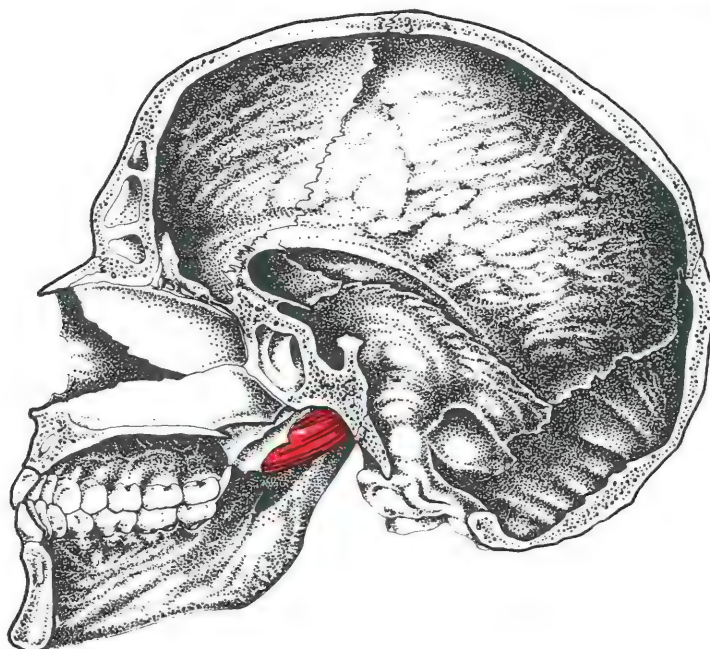
Meridian: Stomach.

Nerve Supply: Lateral pterygoid nerve of the anterior trunk of the mandibular division of the trigeminal nerve.



9—10. External pterygoid.

General Discussion: The external pterygoid muscle can be damaged as a result of a broad palatal tonsillectomy.³⁶ This possibility should especially be considered if there is lack of response in a jaw-opening problem where the mandible deviates laterally. The symphysis menti will swing toward the side of the weak external pterygoid.

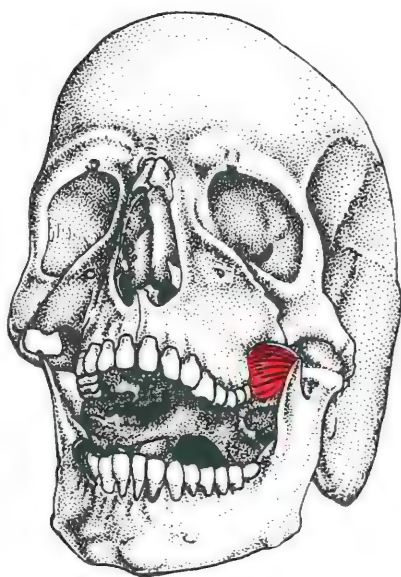


9—11. External pterygoid, sagittal view.

There has been some controversy about the presence of neuromuscular spindle cells in the external pterygoid.^{21, 38} Although some investigators have failed to find them, many others have.⁴⁹ Gill²⁰ found neuromuscular spindle cells primarily in the middle third of human adult external pterygoid muscles.

The lower external pterygoid muscle is the primary muscle in jaw opening. It is usually the muscle involved when a jaw-opening dysfunction is found during an applied kinesiology examination. The muscle is occasionally palpated for tenderness; however, this step is often eliminated because of the difficulty of contacting the muscle. It is necessary to contact the muscle for treatment of muscle proprioceptors when there is a jaw-opening problem.

Contacting the external pterygoid requires a gentle approach. Through the patient's fully opened mouth the physician inserts his finger on the buccal side of the upper molar teeth, past the pterygoid process as far toward the condyle of the mandible as possible. It is rarely possible to feel the usual



9—12. External pterygoid.



9—13. Buccal and inferior approach to palpate the external pterygoid muscle.

Anatomy of Muscles of Mastication, Tongue, and Teeth

indications of swelling and nodules of a suspected neuromuscular spindle cell dysfunction. The usual approach is to make a sweeping digital contact and immediately remove the finger from the mouth. This is a very uncomfortable procedure for the patient. The discomfort is lessened if the physician has the ability to readily direct his pressure to the appropriate point and immediately withdraw his finger.

Nimmo⁴⁸ attempts to contact the superficial section of the external pterygoid externally. The fingers are placed just anterior to the condyle, and

pressure is directed toward the external pterygoid above the mandibular incisure toward the lateral pterygoid muscle. It is recognized that the muscle is deep to this area, but it has been clinically observed that Nimmo's approach is productive of good clinical results in some jaw-opening problems.

Balanced function of this muscle is very important in cases of TMJ dysfunction. The muscle also has a leverage factor into the cranial mechanism, but it does not appear to be as important as the mandibular elevators.

ORGANIZATION OF MUSCLES IN MANDIBULAR MOVEMENT

The major activity of the muscles during various movements can be summarized as follows.

Mandibular elevation: All divisions of the temporalis are active in elevation, as well as the masseter and internal pterygoids.

Jaw opening: External pterygoid and anterior belly of digastric. On forced opening the supra- and infrahyoid muscles are active.

Abduction: Posterior fibers of ipsilateral temporalis; contralateral internal and external pterygoids.

Retraction: Posterior fibers of the temporalis,

and possibly some activity from the deep part of the masseter.

Protraction: Primarily the external pterygoid, with some contribution from the internal pterygoid and the masseter.

Following the anatomy of the teeth and the periodontal ligament, there is a discussion of the neurologic organization of muscles in mandibular movement.

Resting Activity

In a relaxed individual there is no EMG activity of the mandibular elevators, even in the fully upright posture with the lips but not the teeth together.² This total relaxation is ideal when there is no mandibular function; however, the activity of these muscles varies greatly with an individual's activity. For example, when a subject is asked to perform a task, such as pressing a button in response to a lamp being illuminated, stress increases and activity is observed in the masseter muscle.⁶⁰ Other examples are subjects who, while wearing portable biofeedback units, indicated elevated levels of tension in the masseter muscle at various times. The individuals wore the devices from four to seven days and were able to explain the anxiety or emotional reaction that caused the clenching of the teeth. Ten of the fifteen patients showed significant clinical improvement of symptoms associated with clenching simply by recognizing when it was there.⁶⁵

The nocturnal activity of the masseter was plotted by Rugh and Solberg⁶⁵ with portable electromyographic recording equipment worn by a subject during the night. They were able to plot increased bruxism with the subject's heightened emotional states. The increased activity correlated with factors such as a job interview, a fight with father, quitting a

job, an important school exam, etc.

As will be seen later, loss of vertical dimension is an important etiological factor in some temporomandibular joint dysfunction cases. Vertical loss may be caused by constant clenching or bracing. Ricketts⁶⁰ points out that the posterior teeth are designed for intermittent pressures in normal occlusion. When constant pressure is placed on the teeth, as in constant contraction of the mandibular elevators, they can retrude, causing loss of vertical dimension. This is the same basic principle as the constant pressure the orthodontist applies to teeth for change of position. It was previously thought that the posterior teeth did not retrude. It has been known for the past twenty years that any tooth can retrude into the alveolar socket if pressure is constant.

It is necessary to differentially diagnose the cause of increased tension. Correction may require an approach to mental health, occlusion, the cranial primary respiratory mechanism, nutrition, or locally to the muscles. In nearly all cases there will be a primary underlying condition. It may be necessary, however, to also treat secondary factors such as the muscles, especially in a chronic case.

Anatomy of Muscles of Mastication, Tongue, and Teeth

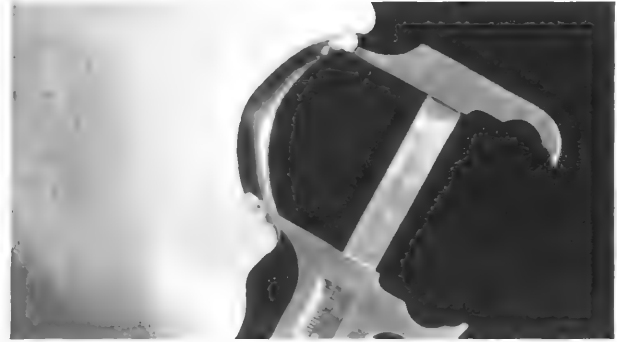
Normal Jaw Aperture

The jaw aperture is measured for interincisal distance with the jaw open as wide as possible. Maximum opening can be expressed in two ways: (1) as the interincisal distance, or (2) as a corrected interincisal distance, which is derived by adding the amount of vertical overlap. Generally, interincisal distance is the figure expressed; it will be used here. There have been many figures quoted in the literature as normal ranges for both men and women. Travell⁷⁰ and Bigelow measured the maximum vertical aperture in fifty young adults and concluded that the normal jaw aperture for men of average height is not less than 50 mm, and for women not less than 45 mm. In a comparison with groups of older men and women, there was essentially the same finding.

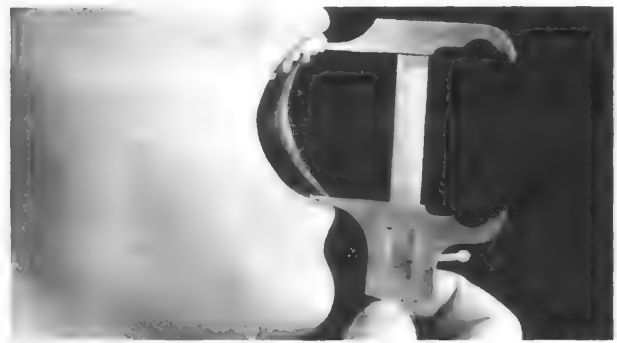
In evaluating the jaw aperture, it is important that the size of an individual be considered. Obviously a smaller individual will have a smaller jaw aperture. Dorrance¹⁴ recommended using the width of the three proximal joints of the phalanges of the 2nd, 3rd and 4th fingers as a guide for the size of the jaw aperture. This provides a guide, regardless of an individual's size. Travell⁷⁰ agrees with this distance. The use of the knuckles on the non-dominant hand closely correlates with the general figures which have been presented.

There has been disagreement in the literature about the value of using the measurement of the maximum vertical opening as a diagnostic index.^{64, 67} Rieder⁶¹ studied 323 adult patients and categorized them as having a history of TMJ dysfunction. This decision was made based on a questionnaire which included indication of "... a history of injury to the TMJ; the presence of crepitus or clicking or popping noises; pain, ringing in the ears, hypermobility, chronic subluxation, spontaneous dislocation, loss of equilibrium, or discomfort from swallowing. . . . The most common symptom described by patients was TMJ noise or pain." Comparison between this group and those who indicated no TMJ disturbance revealed no difference in the range of interincisal opening. He concluded that there was no direct connection between maximum jaw opening and the history of TMJ symptoms. This is in accord with our clinical findings. Some individuals who have severe TMJ dysfunction have an extremely large interincisal opening, while others with the problem have a very limited range of opening. A thorough examination is necessary to determine the functional capability of the masticatory apparatus.

In patients with limited interincisal openings, there is nearly always a clinical hypertonicity of the jaw-closing muscles or failure of proper translation.



9—14. Boley gauge used to measure interincisal distance. The patient's head should be positioned so the Frankfurt line is parallel to the floor.



9—15. Neck and head extension increases the interincisal distance.

(Muscular conditions will be considered in Chapter 14 on "AK Examination and Treatment," and the disc problem will be considered in Chapter 11 on "TMJ Function.")

It remains here to discuss the electromyographic activity of the muscles on maximum jaw opening. The mandibular elevators are basically inactive on jaw opening, but MacDougall and Andrew³⁹ found with maximum jaw opening that the masseter and temporalis muscles have high levels of activity at the terminal part of the movement. This is probably for the protection of the TMJ. Applied kinesiology evaluation may indicate a reactive muscle condition which causes the firing of the temporalis and masseter too early in the jaw-opening phase. This could develop as a result of neuromuscular spindle cell dysfunction in the external pterygoid or possibly some other muscle involved in jaw opening, such as the anterior belly of the digastric. It is necessary to evaluate all the muscles involved in the activity, including those of the supra- and infrahyoid group; they are often responsible for limited jaw opening. The supra- and infrahyoid muscles become active on mouth opening against resistance.

Hyoid Group

The hyoid group of muscles is considered thoroughly in Chapter 13. Here the discussion is limited to its general activity in the stomatognathic system's closed kinematic chain, and activity in mandibular motion.

Part of the anterior muscular chain is made up of the supra- and infrahyoid muscles which stabilize the hyoid to the thorax, skull, and mandible. This portion of the chain is completed by the muscles of mastication connecting the mandible to the skull. In addition, the anterior chain receives contribution from the portion of skeletal muscles which stabilize the head, cervical spine, and thorax, including the sternocleidomastoid and deep cervical flexors.

The principal muscle of mandibular depression or jaw opening is the external pterygoid. Shortly after it fires (1.48 seconds), the anterior belly of the digastric muscle activates. This amount of time diminishes if the head is extended or if the opening is

forced.⁴⁴ While the digastric does not initiate mandibular depression, it plays an important role in regulating the movement. Moyers⁴⁴ examined a patient in which the digastric muscles had been extirpated. While the mandible could be depressed, "... movement was jerky and irregular, particularly in the latter stages of the movement."

When mandibular depression is against resistance, the supra- and infrahyoid muscles as a group become active.³⁹ It appears that in routine opening the only hyoid muscle with major activity is the anterior belly of the digastric.

The mandibular elevators, buccinator, and external pterygoid are the muscles most often involved in muscular problems of TMJ disturbance. When TMJ dysfunction cannot be attributed to those muscles, it is very likely that the hyoid group is involved. Methods of evaluating the hyoid muscles with mandibular movement are discussed in Chapter 14.

Tongue Anatomy

The anatomy and function of the tongue, situated within the curve of the body of the mandible, are very important in considering the function of the stomatognathic system, including the position of the teeth³²

and cranial primary respiratory function. The primary consideration here is the gross appearance, muscles, and nerve supply.

GROSS APPEARANCE

The base, or root, of the tongue is connected to the hyoid bone by the hyoglossi and genioglossi muscles and the hyoglossal membrane. The apex rests against the incisive papilla at the base of the upper incisor teeth. The dorsal or superior surface of the tongue has an oral and a pharyngeal part. There is a median furrow on the oral part, and the entire surface is covered with papillae. In the median plane of the inferior surface of the tongue there is a connection to the floor of the mouth by the frenulum linguae.

Papillae

The papillae are located over the anterior two-thirds of the dorsum of the tongue, giving it a characteristic roughness. The types of papillae are the vallate, fungiformes, filiformes, and simplices. Knowledge of the papillae's appearance is important in nutritional considerations.

Vallate papillae are large and vary from eight to twelve in number. They are located just anterior to the foramen cecum and sulcus terminales, and are shaped like a "V." A papilla is shaped like a truncated

cone, with the broad part projecting a little above the surface of the tongue and having many small, secondary papillae located on the free surface. They are especially numerous within the circular sulcus surrounding the papillae.

Fungiform papillae are more numerous than the vallate and are found chiefly at the sides and apex of the tongue. Usually they are found sparingly over the dorsal surface, but they may sometimes be numerous in that area. The fungiform papillae are large, with rounded eminences, and they have a deep red color. They are covered with secondary papillae.

Filiform papillae are filiform or conical in shape. They cover the anterior two-thirds of the dorsal surface of the tongue and are very small. They are arranged in rows lying parallel to the vallate papillae, except at the apex of the tongue. On their apices are numerous secondary papillae, which are whitish as a result of the thickness and density of the epithelium.

Simplices papillae are similar to the papillae of the skin and cover the entire mucous membrane of the tongue, including the larger papillae.

MUSCLES OF THE TONGUE

The tongue is divided into lateral halves by a septum which is fixed below to the body of the hyoid bone. This gives bilaterality to all the muscles — intrinsic and extrinsic — of the tongue. Extrinsic

muscles include the genioglossus, hyoglossus, styloglossus, chondroglossus, and palatoglossus. The intrinsic muscles are the superior and inferior longitudinal, the transverse, and the vertical.

EXTRINSIC MUSCLES^{33, 47}

Genioglossus

Origin: Upper genial tubercle on the inner surface of the symphysis of the mandible.

Insertion:

Inferior fibers: Into the body of the hyoid bone.

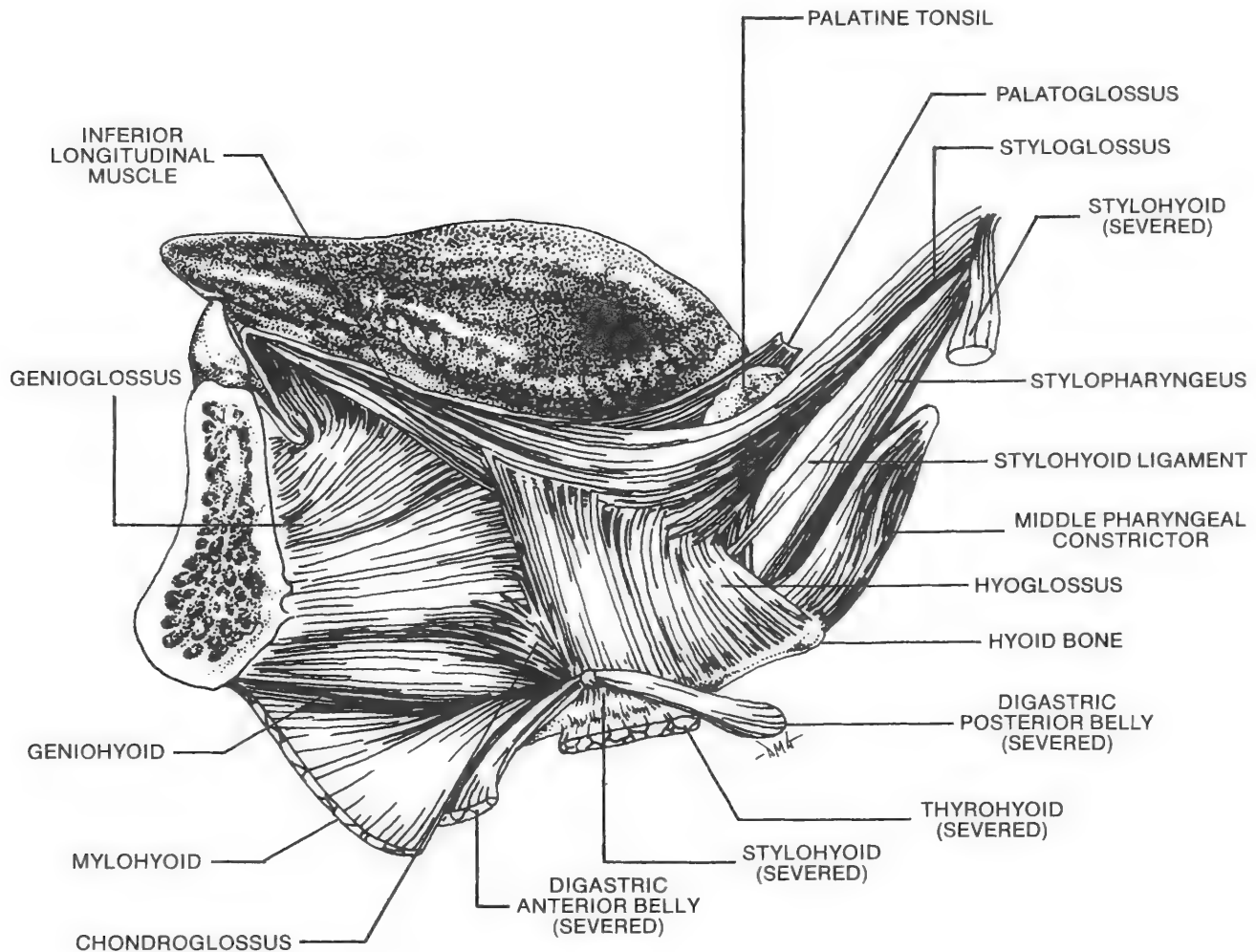
Middle fibers: Into the entire length of the inferior surface of the tongue.

Superior fibers: Into the apex of the tongue.

Action: Inferior and middle fibers draw the tongue forward to protrude its apex from the mouth.

Superior fibers retract the tongue. The muscles, acting bilaterally in their entirety, draw the median part of the tongue down to make the superior surface concave from side to side.

Nerve Supply: Muscular branches of the hypoglossal nerve (cranial XII).



Anatomy of Muscles of Mastication, Tongue, and Teeth

Hyoglossus

Origin: Greater cornu and body of the hyoid bone.

Insertion: Into the side of the tongue between the styloglossus laterally, and medially the inferior longitudinal muscle.

Action: Depresses the sides of the tongue; with the chondroglossus depresses the entire tongue.

Nerve Supply: Muscular branches of the hypoglossal nerve (cranial XII).

Chondroglossus

Origin: Medial side and base of lesser cornu and the body of the hyoid bone.

Insertion: Blends with the intrinsic tongue muscles between the hyoglossus and the genioglossus.

Action: Depresses the tongue.

Nerve Supply: Muscular branches of the hypoglossal nerve (cranial XII).

Styloglossus

Origin: Anterior and lateral surfaces of the styloid process near the apex.

Insertion:

Longitudinal division: Into the side of the tongue near its dorsal surface.

Oblique division: Overlaps the hyoglossus and decussates with its fibers.

Action: Retracts and elevates the tongue.

Nerve Supply: Muscular branches of the hypoglossal nerve (cranial XII).

Palatoglossus

Origin: Anterior surface of the soft palate.

Insertion: Into the side and dorsum of the tongue, blending with the styloglossus and transverse muscle of the tongue.

Action: Lifts the root of the tongue, approximating the palatoglossal arch. With the muscles acting bilaterally, the action closes off the mouth cavity from the oral part of the pharynx.

Nerve Supply: Fibers leaving the medulla in the cranial part of the accessory nerve, reaching the pharyngeal plexus of the vagus nerve.

INTRINSIC MUSCLES

Superior Longitudinal Muscle

Origin: Submucous fibrous layer close to the epiglottis, and from the median fibrous septum.

Insertion: Edge of the tongue.

Action: Shortens and turns the tip and sides of the tongue superiorly when acting bilaterally. Acting unilaterally, it also turns the tip of the tongue to that side.

Nerve Supply: Muscular branches of the hypoglossal nerve (cranial XII).

Inferior Longitudinal Muscle

Origin: From root of tongue, with some fibers from the body of the hyoid bone.

Insertion: Apex of tongue, blending with the fibers of the styloglossus.

Action: When acting bilaterally, shortens the length of the tongue and turns the apex inferiorly and the sides downward. Acting unilaterally, turns the tip of the tongue to that side.

Nerve Supply: Muscular branches of the hypoglossal nerve (cranial XII).

Transverse Muscle

Origin: Median fibrous septum.

Insertion: Submucous fibrous tissue at the sides of the tongue.

Action: Narrows and elongates the tongue.

Nerve Supply: Muscular branches of the hypoglossal nerve (cranial XII).

Vertical Muscle

Origin: Located at the edge of the anterior part of the tongue, with fibers extending from the upper to the under surface.

Insertion: From the dorsal to the ventral surfaces of the tongue.

Action: Flattens and widens the tongue.

Nerve Supply: Muscular branches of the hypoglossal nerve (cranial XII).

Periodontal Ligament^{16, 68, 77}

The periodontal ligament has great importance in the overall consideration of the stomatognathic system. Understanding this structure's role in the system requires a thorough understanding of its histology and its role in the relation of tooth to bone.

The periodontal ligament is composed of fibers, blood vessels, lymphatics, nerves, and cellular elements. It anchors the tooth from its cementum to bone, alveolar tissue, and other teeth. When there is no stress placed on the tooth, the fibers of the periodontal ligament are relaxed and somewhat wavy. When force is applied to the tooth, the tension applied to these relaxed fibers provides a shock absorber mechanism to the attachment of the tooth. There are three types of fibers.⁶⁸

Gingival fibers — connect the cementum to the gingiva.

Transseptal fibers — connect to adjacent teeth.

Alveolar fibers — connect the tooth to the bone.

1. Alveolar crest — These are fibers which

connect the cervical cementum to the lamina dura.

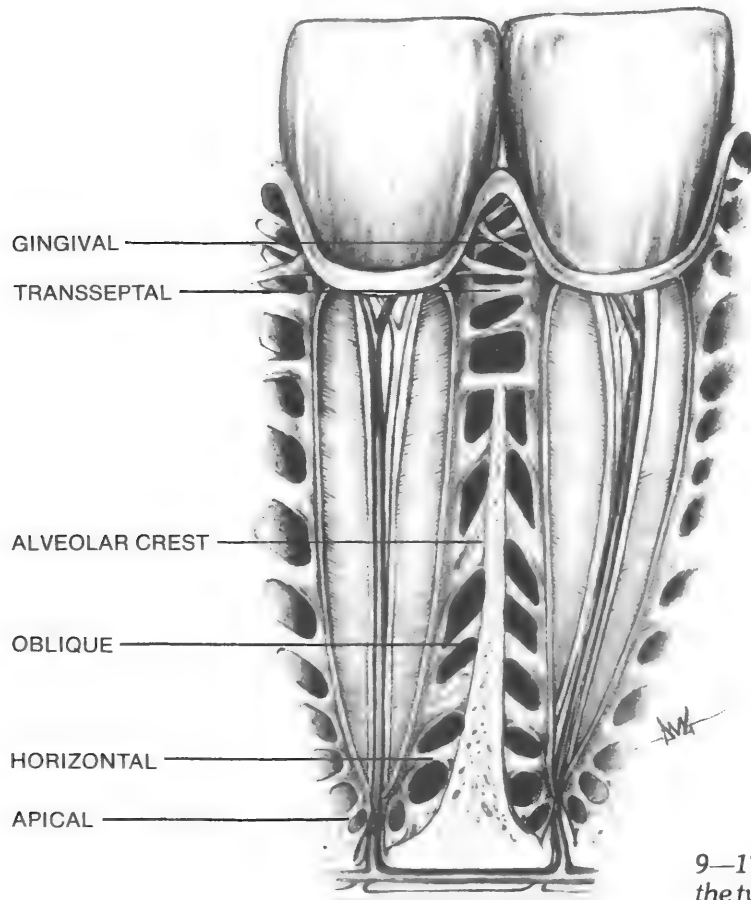
2. Horizontal — Fibers which run at right angles to the long axis of the tooth from cementum to bone.

3. Oblique — Fibers which run from the cementum in an oblique fashion so that they angle from bone to the apex of the tooth. These are the most important and numerous of the periodontal fibers. The oblique arrangement gives additional strength to the tooth. As force is exerted on the tooth toward the apex, the oblique fibers tighten, thus pulling the alveolar socket closer to the tooth.

The periodontal ligaments and associated structure are called the periodontal transmission mechanism, which adapts to the forces applied to the tooth.¹⁵ When function and adaptation of the periodontal transmission mechanism are lost, there will be loss of the tooth.

4. Apical — Fibers from the apical region of the root to the bone.

5. Inter-radicular — Fibers from the crest of the inter-radicular septum to the bifurcation of the roots.



9—17. Enlarged stylized illustration of the types of periodontal ligament fibers.

FUNCTION

The periodontal ligament has four basic functions: (1) supportive, (2) nutritional, (3) formative, and (4) sensory. The supportive function is the mechanical stabilizing effect of the fibers' attachment from the cementum to the tooth's supporting structures. The suspensory arrangement of the ligament cushions and acts as a shock absorber for forces applied to the tooth.

As explained, the alveolar socket is tightened around the tooth when force is applied axially on the tooth. As the tooth is forced into the alveolar socket and the oblique fibers tighten the bone against the tooth, the periodontal space narrows, thus squeezing the blood and lymph vessels and forcing fluids out of the space. As pressure on the tooth is reduced the space re-opens, drawing new fluid into the area; thus a hydraulic piston arrangement supplies nutrition to the area and removes waste products.

The formative function is to repair tissue in the normal renewal process or for pathological reasons. The periodontal ligament is the source of osteoblasts, cementoblasts, fibroblasts, osteoclasts, and phage cells.

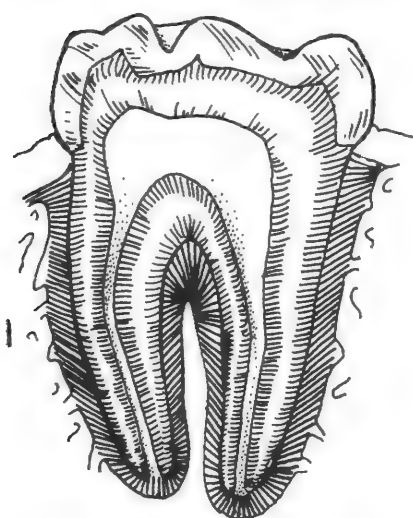
The neurologic function deserves the greatest consideration in our discussion. The far-ranging effects on the nervous system from disturbance in the dentition are unknown. There is evidence that remote problems can be caused directly through the nervous system to areas such as the arm.⁵⁸ The work of Westrum et al.⁷⁶ suggests a cause and effect relationship in alteration of the dentition and changes in the nervous system. These studies may relate only to the pathological conditions of the dentition, or they may expand to functional conditions. In this text

we will deal only with involvements of the stomatognathic system which are solidly established in the literature and relate with the interactions of the stomatognathic system and its interaction with the total body on a functional basis.

The nerve receptors of the periodontal ligament are nociceptors and proprioceptors. The proprioceptors are sensitive enough to determine the thinnest piece of paper placed between the teeth. It is through this mechanism that malocclusion is sensed, which may cause the entire stomatognathic system to change. The least amount of malocclusion — which may only be a very slight contact by a single tooth in advance of the others — is perceived and integrated into muscular activity. This is to change the muscular pattern and avoid premature contact, saving wear and tear on that tooth; this is an ideal situation. Sometimes the muscles cannot find a mandibular position free of prematurities.

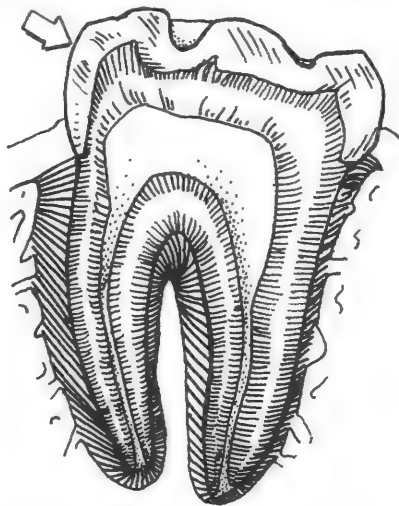
Another mechanism protective of the teeth is reflex from the nociceptor. An example of this action is when one is eating cherries and bites on a stone. There is immediate reflex opening of the jaw to avoid injury to the teeth.⁵⁷

The opening reflex is also present in normal chewing. There is a difference in the muscular activity between those with natural dentition and those with dentures.³⁶ The jaw-closing muscles are completely depressed during mouth opening in the presence of natural dentition. These reciprocal relationships are not as evident in denture wearers. This fine regulation of mastication is from the feedback control of the afferent system to regulate the efferent activity causing mandibular motion.

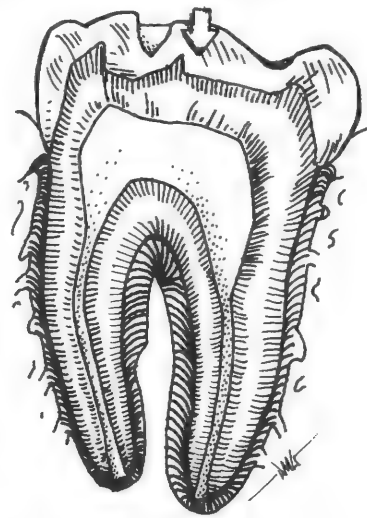


Relaxed periodontal ligament.

9—18.



Force applied which is somewhat absorbed by the periodontal ligaments.



Axial force tightens the bone around the roots because of the angle of the oblique fibers.

Anatomy of Muscles of Mastication, Tongue, and Teeth

Neuroanatomic Basis of Mandibular Movement

The importance of the proprioceptive system in integrating the body's adaptive mechanism for posture, locomotion, etc., is emphasized throughout applied kinesiology. There are many examination procedures and therapeutic approaches based on this mechanism. Activity within the stomatognathic system initiated by the proprioceptors in the periodontal ligament is much the same as with proprioceptive function and dysfunction in other areas of the body. A proprioceptor signals the status of position, stress, etc., through the afferent system. If necessary, a reaction to the changing conditions is sent over the efferent system.

The nociceptors of the periodontal ligament send information about the area's environment over the afferent system. Stimulation to these receptors may lead to conscious and/or reflex reaction. In a study which produced noxious stimuli to a tooth to create pain, Robertson et al.⁶² found that pain was radiated to numerous areas, particularly that of cranial nerve V. They also found that pain developed in the neck along with suboccipital headaches.

Corbin and Harrison¹¹ found that the reflex control of mastication is primarily the function of the mesencephalic nucleus of the trigeminal nerve. Proprioceptors from the teeth, muscles, and temporomandibular joint contribute afferent supply to the motor nucleus of the trigeminal nerve, and also to higher centers to moderate volitional control.³⁶

The normal jaw-opening reflex is initiated by stimulation of the receptors in the periodontal ligament.^{2, 29, 46, 59} This reflex is an inhibition of the mandibular elevators after tooth contact sufficient to stimulate the receptors in the periodontal ligament.⁵⁵ Beaudreau et al.³ remarked in studying this reflex that it was interesting how light a tap on the tooth was required to initiate inhibition. Inhibition is observed electromyographically by a silent period (no electrical activity) of the mandibular elevators, which is initiated after a mean interval of 10-14 msec after initial contact. The inhibition lasts a mean duration of 12-20 msec. In addition to inhibition of the mandibular elevators, activity develops in the anterior belly of the digastric but not in the external pterygoid. This reflex apparently is to protect the teeth in a manner similar to that in which a Golgi tendon organ protects a muscle from overcontraction. If, in fact, there is no opening of the jaw from the inhibition of the mandibular elevators and facilitation of the anterior belly of the digastric due to the short time involved, there is at least a reduction of force.²⁹ No studies have been found regarding the activity taking place during persistent tooth contact.

It is apparent from a study done by Hannam and Matthews³¹ on adult cats that the reflex is initiated from the proprioceptors in the periodontal ligament. It was observed there was no decrease in the jaw-opening reflex when the gingiva around the tooth was removed, nor when the tooth pulp was extirpated. The reflex disappeared on infiltration of lignocaine over the root of the tooth. Their study was supported by Anderson and Mahan,¹ who implanted electrodes in the periodontal ligament and demonstrated vigorous jaw depression activity on electrical stimulation. The reflex remained after the pulpal nerve was removed by endodontal therapy, revealing it was not involved in the jaw depression reflex.

Measurement of the maximum bite force of individual teeth reveals a difference from tooth to tooth.³⁴ This appears to be due to the limitation of bite force controlled by muscle inhibition as a result of stimulation to the periodontal ligament proprioceptors.

Perry⁵⁴ proposed a hypothesis which is now generally accepted for the neuroanatomic basis of mandibular movement. There is considerable electromyographic evidence that this hypothesis of mandibular movement is correct. It suggests that mandibular motion is specifically guided by neuromuscular interaction to dictate the final closing position of the mandible toward intercuspation. A premature tooth contact (prematurity) can develop as a result of ill-fitting restorations or prostheses, or direct trauma to the tooth. Malocclusion can also develop as a result of a cranial fault, which can disturb the relationship of the right maxilla with the left, or temporal bone rotation can change the mandible's position. The prematurity stimulates the periodontal ligament to send "... sensory signals into a reflex system which will guide the mandible by means of its musculature away from the areas of noxious, premature contacts. This musculature is so positioned anatomically that movements in a guiding fashion can be instituted in both the lateral and sagittal plane."

Whether one opens and closes the jaw slowly or rapidly, the final tooth contact is not just by happenstance of the so-called hinge axis of the mandible.⁷⁵ The closing activity is a pre-programmed, neuromuscular activity that takes place as a subconsciously learned response. The finger-to-nose neurological test is an example of the proprioceptive feedback mechanism present in the activity of the mandibular positioning reflexes. When the nervous system is functioning normally, an individual with eyes closed can easily place his finger on his nose. In the mandibular closing reflexes, the initial information

comes from the proprioceptors monitoring tooth contact. When dentition is normal, intercuspation develops with no premature or initial contacts and consequent sliding. Shore⁶⁸ states that in normal activity " . . . the condyles in the overwhelming majority of individuals are slightly in front of their 'most retruded' position; in other words the capsule is not in a state of unique tension but is in all its parts more or less relaxed. This in turn must be interpreted as ruling out proprioceptors of the capsule as directing the automatic closing of the jaws."

Stimulation of the periodontal proprioceptors produces a quantity of afferent information which is interpreted to efferently control the muscles involved in mandibular closing. This extreme quantity of information must be properly processed for optimum function. If there is no malocclusion, the act of stimulating the proprioceptors and the following interpretation and control of muscles is repeated over and over, and an engram develops which becomes the mandible's stable closing pattern. To an individual who has studied the interaction of the muscles of mastication, it should be amazing that a person can snap his jaw closed from a fully opened position and have perfect coordination toward complete intercuspation. Commenting on the wide-open jaw snapping shut with the muscles moving the mandible swiftly and unerringly into the centric occlusal position, DuBrul¹⁶ makes the colorful statement, "This is perhaps an extreme example of the nicety of coordination in all normal bodily movements." As with any other activity in the nervous system, the engram is dynamic; if there is a slight change in the dentition over a period of time, the engram adapts to it. If, however, there is a rapid change in the dentition from a cranial fault, dental procedure, or trauma to the tooth, the engram cannot change rapidly enough to meet the immediate demand, and malocclusion develops.

If there are many prematurities or severe malocclusion, afferent signals from the periodontal ligaments send information that cannot be processed because there is no position that muscle interaction can produce for a good occlusion.⁵² The resulting activity will probably be toward the optimum occlusion available under the adverse conditions.

A very interesting study was done by Munro.⁴⁵ It included individuals who were considered "potentially pathological" regarding TMJ dysfunction. This was determined by observing clicking and crepitus in the articulation, as well as symptoms which could possibly refer to the TMJ, such as headaches, facial pain, etc. The study was an outgrowth of another study where a microphone was placed over the zygoma to record tooth contact on jaw closure,

ultimately evaluating the silent period of the jaw-closing muscles. The group considered here was excluded from the original study because of the symptomatic complex and TMJ clicking or crepitus observed on the microphone. In this group the muscular action, as well as the silent period, varied from the larger group which was considered normal. Prematurities were sometimes observed by double sounds picked up by the microphone. There was usually inhibition of the muscle contraction (silent period) following the prematurity. Occasionally there were two successive inhibitions of the jaw-closing muscles after the double contact. Sometimes there was activity of the masseter and anterior temporalis in the opening phase of the cycle; in other words, the closing muscles were active during jaw opening. In over half the group — including those with prematurities — there was no silent period, showing a failure of muscle inhibition on tooth contact as is normal. This happened more often in the anterior temporalis than in the masseter muscle. Of very great importance in this study is the high incidence (55%) of individuals having disorganization of the muscles in a group which was only "potentially pathological." The incidence of muscular incoordination and improper timing of the silent period was as high in this group as those in an earlier study by Griffin and Munro³⁰ where the group had definite temporomandibular joint dysfunction. This fact, combined with minimal evidence of pain or other dysfunction as observed by the subject, indicates that there is probability of dysfunction for a considerable time before actual pathology develops.

Another study giving evidence that improper stimulation or disturbance of the proprioceptors in the periodontal ligaments disturbs function in the muscles of mastication is presented by Bessette et al.⁶ A control group with no evidence of malocclusion or other dental pathology was compared with a group seeking treatment for one, or a combination of, the following complaints: (1) pain in one or both temporomandibular joints, (2) deviation or limitation in opening the mouth, and (3) presence of TMJ sounds such as clicking or cracking in the intermediate ranges of mandibular movement. They found in the group with the "TMJ syndrome" that there was an abnormally long silent period following elicitation of a jaw-jerk reflex. Conventional occlusal adjustment returned the silent periods to normal in all but two individuals. In these two the therapy also failed to relieve the symptoms. The study suggested that the duration of the silent period could be a useful diagnostic tool and an aid in the prognosis and treatment of "TMJ syndrome."

The silent period is also shown by Widmalm⁷⁸ to

be extended after a jaw-jerk reflex. Among other factors he considered as a possible cause of, and factors influencing, the reflex response are several aspects which correlate with the applied kinesiology hypothesis of temporomandibular joint dysfunction: (1) vibrations elicited by tapping the chin, exciting muscle spindles or periodontal receptors; (2) disfacilitation after the initial excitation (unloading); and (3) inhibition from Golgi tendon organs. These all relate to the proprioceptors often treated by applied kinesiology to relieve temporomandibular joint dysfunction.

To summarize the neuromuscular control of mandibular movement, it is important to consider the afferent-to-efferent control from the periodontal ligament to the muscles of mastication. This pattern develops into an engram which is dynamic and can slowly adapt to changing occlusion but cannot adapt to severe malocclusion. Carlsöö⁹ observed on electromyography that the muscular patterns varied with the type of bite. In the same study, he thoroughly evaluated the moments of force of the mandibular elevators in comparing muscle action evaluated with EMG. He stated, "Notwithstanding that the mechanical qualifications of the different muscles are the same, on the whole, from one test subject to another, it is shown that the formation of the innervation pattern appears to be intimately related to the bite type." Malocclusion stimulation to the periodontal ligament proprioceptors causes the rest position to be unstable. Eliminating the improper stimulation to the proprioceptors causes the rest position to then become stable.⁵³

This discussion has related primarily to stimulation of the periodontal ligament proprioceptors as the entrance into the afferent system to create imbalance of the muscles of mastication. This muscle imbalance can pull into the cranium to possibly cause

cranial faults. A different origin of the disturbance can be muscle pain created by any of many factors. Pain induces a sustained reflex contraction of the jaw muscle, with increased pain and further contraction. The imbalanced muscular function may cause malocclusion, which in turn stimulates the periodontal ligament proprioceptors and throws the system further off-balance.³⁶

Our primary concern in evaluating the masticatory muscles of the stomatognathic system is whether the organization of the muscles is appropriate for the body's needs. If, due to a prematurity or some other factor, the proprioceptors in the periodontal ligament are stimulated to create imbalance of the muscles of mastication, there will be stress on the cranium in such a way that the cranial primary respiratory mechanism is disturbed. Neurologic disorganization may also be introduced into the nervous system. Neurologic disorganization throughout the body as a result of improper function of the masticatory muscles is probably the most deleterious factor to health caused by dysfunction in this area. Perry,⁵⁴ in an electromyographic study of patients with temporomandibular joint dysfunction, observed some individuals who complained of a dull, aching pain in the dorsum of the neck and out into the posterior of the shoulder. Electromyography indicated abnormal electrical activity of the muscles in these regions. Since the upper trapezius receives part of its nerve supply from cranial XI, it is probable that the disturbance was from cranial primary respiratory dysfunction. Perry explains the problem as a disturbance in the closed muscular kinematic chain of the stomatognathic system, previously described. He points out, "The anatomical division of muscles into groups is necessary in teaching and understanding the function of the single unit, but the function of the whole is what the structures are there for."

Prodigious Nerve Supply

The study of the proprioceptors of the periodontal ligament touches on one aspect of the prodigious nerve supply to the stomatognathic area. Many investigators^{19, 25, 36, 40} have pointed to the large area in the cerebral cortex devoted to the stomatognathic area as plotted by Penfield and Rasmussen.⁵¹ The homuncular representation which illustrates their work shows a very large jaw and face while other portions of the body, with the exception of the hand, are relatively small.

Indeed the stomatognathic area has a very prodigious nerve supply, both efferent and afferent.

There is sound neurological evidence in the literature that provides substantial basis for the examination procedures, therapeutic efforts, and clinical results associated with correction of the stomatognathic system. Some of these findings have been referred to in the preceding material on the proprioceptors of the periodontal ligament; other material is referenced throughout this text. Even with this, there are clinical observations that cannot be understood with the current data base available in neurophysiology and anatomy. One such observation is the clinical association of various areas on the dental arches with

Anatomy of Muscles of Mastication, Tongue, and Teeth

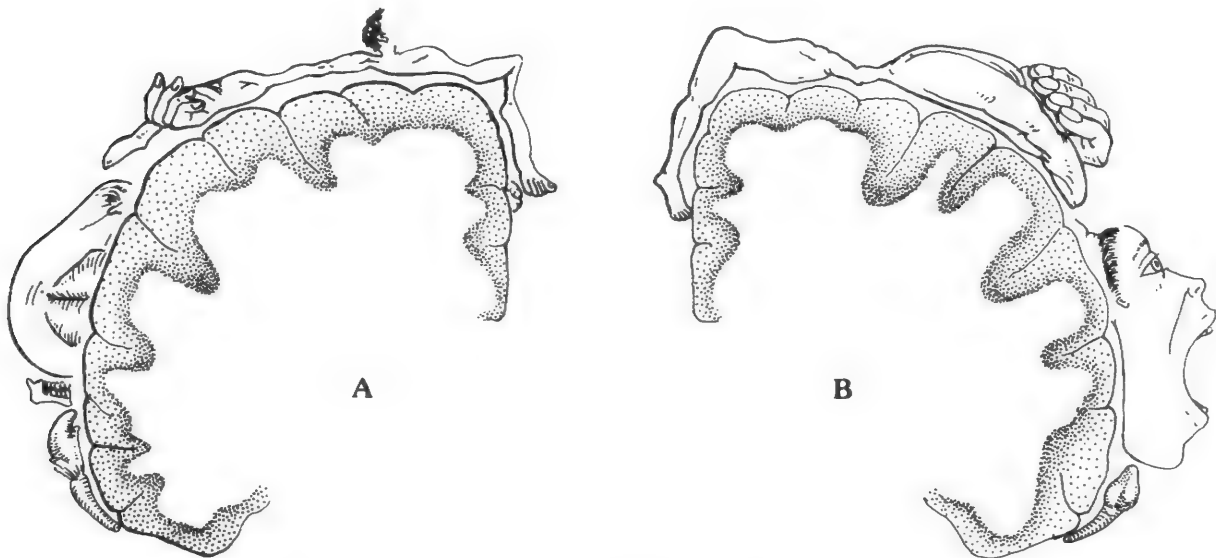
specific muscles and organs or glands of the body. This association is similar to the organ-muscle association which has developed on a clinical basis in applied kinesiology. The association is not a factor to weigh heavily in a final diagnosis; it is, rather, an observation that should lead to further evaluation with standard diagnostic procedures. At this stage of our understanding it is only a body language factor which helps guide the investigation; it is not a hard-core rule of association. To help put this in perspective, it has long been noted that if a physician observes pallor in an individual there is a possibility of anemia. Further indication would be a lack of redness on the internal lower eyelid. Even though this is good body language that suggests further evaluation for anemia, no physician would be justified in making that diagnosis without a blood count to quantify it.

Observation of disturbed areas in the dentition led Goodheart²⁶ to correlate specific areas of the dental arches with apparent organ or gland dysfunction and its associated muscular involvement. The relation is homuncular, relating with the temporal spenoidal (TS) line (see Volume I of this series). The TS line is represented in the lower dental arch beginning with the third molar, associated with the psoas, and progressing around to the central incisor, associated with the gluteus medius. The TS line representation continues in the upper dental arch with the central incisor representing the neck flexors and extensors, proceeding to the third molar, representing the trapezius (middle division). There is often a correlation between positive therapy localization

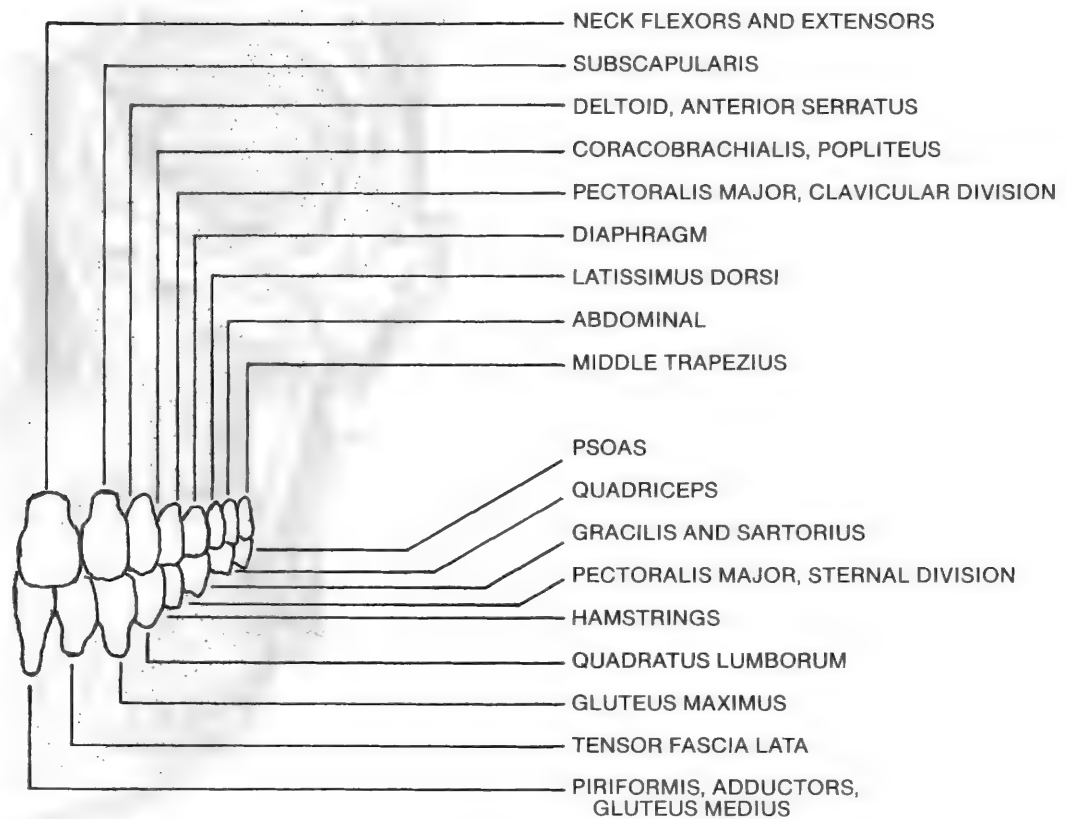
and palpation on the TS line with positive therapy localization and neurologic challenge on the corresponding dental arch area. The representation in the dental arch is not on an exact tooth-for-tooth basis, but is rather in a general area. The accompanying chart represents the approximate location, but the actual indication of involvement may be found on an adjacent tooth.

This clinical relationship has been taught to many dentists in classes presented by diplomates of ICAK, and also by Eversaul.¹⁸ It has been reported that there is often a clinical disturbance of a localized area in the dental arch which may be endodontal, periodontal, or pain of unknown origin that fails to respond to treatment. On further investigation it may be found that the patient has some functional or pathological disturbance in the muscle, organ, or gland associated with that dental arch area. Correction of the remote problem yields improvement in the dental area. No studies of this relationship have been reported in the literature.

It has also been clinically observed that sometimes a specific muscle which tests weak or hypertonic on manual muscle testing cannot be corrected until attention is given to a tooth which correlates with the homuncular representation for that muscle. It appears the correlation is also applicable in reverse. There may be an organ or muscle dysfunction primary to a diseased tooth or its periodontal tissue. Correction of the remote factor disturbing the tooth or tissue appears to improve function of the dental involvement.



9—19. Sensory (A) and motor (B) homunculus. After Penfield and Rasmussen,⁵¹ *Cerebral Cortex of Man*, The Macmillan Co.



9—20. Temporal sphenoidal line homuncular representation in the dental arches.

Challenge and Manipulation of Teeth

Tooth disturbance can be a result of malocclusion, which is discussed in Chapter 12. In some instances there is no evidence during occlusal evaluation why a tooth will show positive therapy localization. There are two possibilities: (1) there is pathology of the tooth or periodontal tissues, or (2) there is a disturbance of the receptors in the periodontal ligament. Clinical evidence indicates that the periodontal ligament can be disturbed in such a way that it produces afferent impulses which are inappropriate to the status of the tooth. It may be that the tooth has been malposed in the alveolar socket, possibly from some destructive force during mastication. The working hypothesis is that the periodontal ligament is traumatized, possibly with adhesions which interfere with its normal function. The objective of evaluation and treatment is to return the tooth and its periodontal ligament receptors to normal function. To determine receptor dysfunction, the tooth can be challenged. The procedure is to press on the tooth with a specific vector of force and test a previously strong indicator muscle for weaken-

ing. If positive, it is presumed that the proprioceptors or nociceptors are being stimulated in an adverse manner, creating temporary disorganization in the neuronal pools, and the indicator muscle fails to adapt to the changing pressure on the tooth from the examiner's test. The indicator muscle will weaken for several seconds, or for a considerable length of time in some cases. In any event, it always weakens long enough for the examiner to make the evaluation. The vector of force which gives a positive response can be in any direction. It may be buccal, lingual, mesial, distal, or toward the apex of the tooth. Occasionally it may even be traction on the tooth that causes the positive indication. After the physician finds the positive vector of force and an indicator muscle weakens, the patient is requested to take a phase of respiration, usually inspiration. The indicator muscle is again tested to determine if the phase of respiration caused it to regain its strength. If inspiration did not abolish the positive challenge, then expiration will. Another way to observe the challenge-respiration correlation is to have a patient take a phase of

Anatomy of Muscles of Mastication, Tongue, and Teeth

respiration while applying the vector of force previously found to be positive. When the correct phase of respiration is combined with the previously positive challenge vector, there will be no weakening of an indicator muscle.

Correction of disturbance in the tooth's neurologic function is obtained by manipulation of the tooth with the appropriate respiration. The tooth is pressed with three or four pounds of pressure in the direction of positive challenge while the patient takes the phase of respiration which abolished the challenge. This is repeated four or five times; then the tooth is re-evaluated by challenge and therapy localization to determine the effectiveness of the correction. Interestingly, in edentulous individuals the homuncular representation still appears to be present in the dental arches. It does not appear to be as important as when the teeth are present; however, it may occasionally need evaluation and treatment. The procedure is basically the same. The gingiva is challenged in various vectors and the phase of respiration is found which abolishes the positive challenge. Treat in the same manner as for a neurologic tooth.

There are many indications to evaluate for a neurologic tooth. Blaich⁷ uses a screening procedure after other obvious findings have been corrected. The patient is asked to bite down gently and a previously strong indicator muscle is tested for weakening. If positive, the possibility of neurologic tooth involvement is then considered. The usual examination and correction procedures described above are used.

Testing a strong indicator muscle while the patient bites down as described above may show no weakening of the muscle; the same test done immediately after correcting cranial faults may then be positive. There are several possible reasons for this change. One is structural, where the cranial correction has altered the relation of the maxillae to each other, changing the shape of the upper arch, or the mandible is shifted in its relation to the upper dental arch. The mandible can potentially change as a result of counter-rotation of the temporal bones, which is often found when there are inspiration and expiration assist cranial faults on opposite sides. Not only is the mandibular fossa changed, but the origins of the muscles are changed very slightly. This immediate change of occlusion does not now fit the engram of muscular contraction which has developed to control mandibular movement into optimum intercuspation. We are reminded of Dawson's¹³ comment of how little occlusal disrelation is necessary to cause problems.

Another reason correction of cranial faults may

change occlusion is that there may have been peripheral nerve entrapment of cranial V, causing improper control of the masticatory muscles. Their return to normal immediately changes the occlusion; again, the engram for mandibular closing is no longer correct for the present circumstances.

Another possibility is that the malocclusion resulting from cranial correction causes a return of the cranial faults just corrected. Clenching the teeth causes the mandibular elevators to reverse origin and insertion. With the mandible as the stable origin, the movable part is the cranium; it distorts back to the cranial fault stage to match the occlusion.

Organization of the occlusion with the cranial primary respiratory system will be discussed in detail later; now it is important to consider the role of the neurologic tooth in this relationship. If, after correcting a cranial fault, there is weakening of a previously strong indicator muscle as the patient bites, evaluate for neurologic tooth by therapy localization and challenge. If present, its correction may eliminate weakening of the indicator muscle. It may also eliminate the return of the cranial fault, if one was present. Apparently, manipulation of the tooth re-organizes the proprioceptors in the periodontal ligament, thus favorably influencing the engram controlling organization of the muscles of mastication.

Another indication to evaluate for a neurologic tooth is localized pain in the dentition. Apparently there are some cases where nociceptors in the periodontal ligament are improperly stimulated and can be returned to normal by manipulation of the tooth as described. Of course, it is necessary to make the usual differential diagnosis for possible pathology.³⁵

Return of a positive neurologic tooth indicates a need to evaluate for malocclusion (discussed in Chapter 12) and possible disturbance in the clinically associated muscle, organ, or gland. For example, if the lower first molar is recurrently disturbed, the problem may be associated with stress of the gracilis and sartorius. These muscles provide specific support to the pelvis. When involved they often relate to a pelvic disturbance where the ilium's posterior superior iliac spine has moved posteriorly and inferiorly. Correction of the structural disturbance in the pelvis and attention to the sartorius or gracilis muscle for return of normal strength may eliminate the recurrent neurologic tooth.

Another clinical relationship is the adrenal gland with the sartorius, gracilis, and lower first molar. An individual may have relative hypoadrenia for any of the many reasons discussed in Volume V. Nutritional and other approaches in applied kinesiology may be necessary to return the adrenal to normal function.

Its improvement is often reflected in elimination of the recurrent neurologic tooth.

The above interactions are all two-way streets. The recurrent neurologic tooth can be a result of structure, organ, or gland disturbance; continued involvement of the structure, organ, or gland can be a result of the neurologic tooth. A cause-effect relationship can often be established by using two-handed therapy localization. This is where one point shows a positive therapy localization, but it is cancelled by another. For example, if the tooth therapy localizes but the positive indication is

eliminated by touching a reflex area to an organ or a muscle, there is evidence that the second point therapy localized is adversely influencing the first point. This is discussed more thoroughly in Volume I.

In the event of a recurrent neurologic tooth with none of the previously described factors present, there is a possibility zinc is needed. Goodheart²⁶ clinically observed that if zinc were administered orally, it often eliminated the recurrent neurologic tooth. The best form of zinc appears to be found in small quantities along with other nutritional supplements, rather than a zinc supplement by itself.

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Chapter 10

Cranial and Stomatognathic Area Interdependence

Reference has been made periodically in this text to the interaction of the stomatognathic area with the cranial primary respiratory system. It is the purpose of this chapter to emphasize the importance of this interaction. Sometimes the stomatognathic area is examined without knowledge of or attention to the cranial primary respiratory mechanism; the converse is also true, and effort is made to correct cranial dysfunction without attention to the stomatognathic area. The educational process tends to break down body activities into sections for convenient study. This is adequate if the delineation stops there and does not extend into areas of therapeutic specialization. Trouble may develop when a specialist devotes extensive study and practice to very intricate and technical examination and therapeutic approaches, but remains unaware of how other areas may influence his specialization.

An excellent example is a dentist who specializes in equilibration. This example is chosen because of the fine detail to which many systems of equilibration have been refined. Often the art and science of equilibration deals with relating restorations, such as a crown, to a patient's natural dentition. This is fine; it is imperative that it be done correctly. In this case the restoration is a known factor creating the need for equilibration. When the prosthesis is equilibrated to the natural dentition all is well, with the system functioning as it should. Unfortunately, there are times when equilibration is done but the etiology of the malocclusion is less than clear. In a study of 277 patients with temporomandibular joint disturbance, 52% revealed no known etiology.²⁰ Actually there is always a reason for any disturbance in the body; the problem is that sometimes we as physicians do not

recognize it. The basic underlying cause of a problem may be in an area of expertise that is foreign to the examining physician. A fundamental rule in diagnosis is that you cannot recognize what you do not know. In the example given, it may be that the patient developed a disturbance in the cranial primary respiratory system for any of many reasons — a blow to the head, structural imbalance lower in the body, or from chemical or mental factors. The important point is that the patient does not understand the cranial disturbance or, if he is aware of it, does not tell the dentist about the blow on the head, injury to the ankle, or whatever. Patients tell their doctors what they feel the doctors need to know. An injury to the ankle does not seem like relevant information for a dentist; in fact, unless a dentist has been trained in the total body concept, that recent injury or other factor would not seem pertinent. The malocclusion present may be a direct result of a cranial disturbance. Correction of the cranium would immediately return the occlusion to normal with no equilibration necessary, if the condition is not chronic. Not recognizing this, the dentist may do a fine job of equilibrating the patient's occlusion under the circumstances. Unfortunately, there is now an excellent occlusion to an imbalanced cranium. The patient may then go to another doctor, seeking relief for some apparently unassociated symptom which may be in the foot, lower back, head, or whatever. Correction returns the cranial primary respiratory mechanism to normal function, but now there is a malocclusion because of the occlusal equilibration made when the cranium was distorted. The malocclusion may cause the cranial correction not to hold and the two doctors, oblivious of each other's treatments,

Cranial and Stomatognathic Area Interdependence

do not know why their therapeutic efforts are failing.

It isn't even necessary for the patient to be seeing another doctor at the same time. Since the body is a self-correcting, self-maintaining mechanism, a cranial fault that develops as a result of a blow on the head will often be corrected by the body's natural efforts. Some of the mechanisms by which the body returns itself to normal will become clear as more body interactions are presented throughout this volume.

Not only does the stomatognathic area relate with the cranial primary respiratory mechanism, it is in fact part of it. This is the reason Shore's⁴⁷ defining of the stomatognathic system to include basically all structures and tissues above the shoulder girdle is so valuable. As has been mentioned in these writings, the interaction does not stop even at the shoulder girdle. The body functions as a total, integrated whole. Integration of the mandible with cranial-sacral primary respiratory motion can be observed very easily by extending the evaluation procedures used

to determine the type of cranial motion explained in Chapter 3, page 87. This procedure simply uses the examiner's digital pressure to resist the normal motion of skull bones while the patient takes a phase of respiration. A previously strong indicator muscle will weaken if the examiner resists the motion desired by the body. If the examiner contacts the mandible bilaterally on the posterior borders of the rami and gently presses the mandible in an anterior direction while the patient takes a deep inspiration, a previously strong indicator muscle will weaken¹⁷ if the patient is functioning normally. This indicates that the mandible moves somewhat posteriorly on inspiration. This microphysiologic motion appears to be very important in normal harmony of body function. Additionally, the gross structure of the mandible and its muscle balance must be in harmony with the gross cranial structure, as well as its microphysiologic function.

Mandibular Influence on Cranial Function

The influence of mandibular motion on the cranium can be easily observed. Its activity was uniquely described by W.B. May,³⁵ a dentist who did much to promote the importance of mandibular function with total health problems. He stated, "If the cranium is a pump for cerebrospinal fluid, then the mandible is the pump handle." It does appear that valuable motion and equalizing forces are normally directed into the cranium by mandibular action.

This interaction can readily be observed in an individual who has cranial faults. First, examine the skull using the methods presented in the first section of this text. Record all cranial faults for re-evaluation, but do not treat them. Have the subject stretch his jaw open to its maximum and then close, not allowing the teeth to make contact. Repeat this opening and closing procedure six or more times, and then re-examine the skull for cranial faults. In most instances all the cranial faults will be temporarily gone. In some very rigid skulls with severely locked cranial faults, it may be necessary to have the subject stretch his jaw open as many as fifty times. There is the rare individual who has such severe, locked-in cranial faults that this procedure will not even temporarily improve function. In most cases, the apparently improved cranial function will last long enough for the examiner to re-evaluate the skull. In individuals who have only moderate cranial faults, the improvement will last longer; its duration will be shorter in

those with severe faults. In some people with only minimal cranial faults, this activity may make a permanent correction. In this case the system would probably have corrected itself anyway.

Silverman's⁴⁸ study on how skull distortion affects occlusion is discussed in Chapter 2. He observed that imbalanced pressure on the skull from various sleeping postures disturbed a normal occlusion. When the occlusion was good, quickly closing the mandible produced a staccato-like sound on intercuspation. If there was malocclusion with prematurity, a deflective occlusal contact was evident from the change of sound. This change of sound can be objectively recorded.⁵⁰ Further investigation revealed that digital pressure on the skull in various areas changed a normal occlusion to one of prematurity. Time alone would return the malocclusion to normal, but he found that it could be returned much more rapidly by swallowing several times. In all cases the occlusion returned to normal after eating breakfast. Further investigation finally revealed that taking a deep inspiration would return normal occlusion most rapidly. From these observations Silverman recommended a procedure for dentists to use prior to equilibration to make certain the skull was normally balanced. This is discussed further in Chapter 12 on "Occlusion."

The swallowing mechanism is similar to the wide mandibular opening previously described. In some

cases of minimal cranial faults, simply swallowing several times will temporarily eliminate evidence of cranial faults. Swallowing activates some of the hyoid muscles; more important in this consideration, it activates the mandibular closing muscles as the teeth are brought into intercuspation and the jaw is stabilized for the hyoid activity in swallowing. Tongue motion also influences the cranium.

If the occlusion, muscles of mastication, and cranium are not functioning harmoniously, swallowing will not improve cranial activity. In fact, it may produce evidence of subclinical faults which were not evident during the initial evaluation.

Temporarily eliminating evidence of cranial faults by stretching the jaw open several times or swallowing is interesting, but what does it mean? It shows the influence of the masticatory muscles on cranial primary respiratory function. When the mandible is opened to its maximum, the mandibular closers pull strongly on the skull. The masseter pulls on the zygomatic arch, with considerable leverage on the temporal bone, and the maxilla is moved by way of the zygomatic bone. The internal pterygoid pulls on the lateral pterygoid plate and pyramidal process of the sphenoid and palatine bones. The temporalis pulls on the parietal, frontal, temporal, and sphenoid bones by way of the temporal fossa.

There is additional activity besides the traction

on the bones from the muscles stretching to their maximum. Electromyographic evidence reveals that the masseter and temporalis become considerably active on maximal mouth opening;³¹ thus they are actively pulling on what are ordinarily their origins. In addition, the major jaw opener — the external pterygoid — acts to mobilize the skull. The action of the external pterygoid at its limit of motion is pulling on the greater wing of the sphenoid and the pterygoid process. All this activates the closed kinematic chain of the cranium, stimulating physiological motion. Often the introduction of force which creates motion in the skull is adequate for correcting minimal cranial faults. On these occasions the increased motion unlocks the mechanism, allowing it to return to its normal autonomous motion.

The philosophy of applied kinesiology recognizes that nothing occurs in the body as random activity. For every body action there is a purpose — improved function. Granted, sometimes the actions are compensating for some form of dysfunction. With the thought that no function taking place in the body is random, is it possible that when an individual yawns very widely it is an effort of the body to activate the cranial primary respiratory mechanism? In any event, it can readily be seen that forced mouth opening influences the mechanism.

NORMAL MASTICATORY ACTION

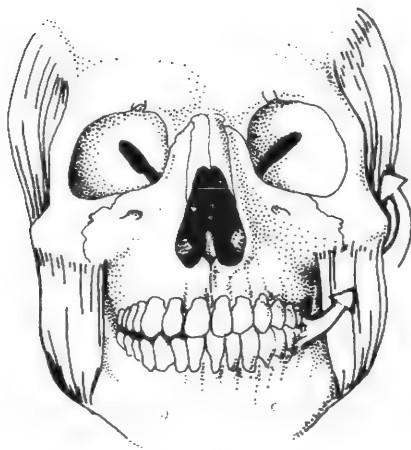
When there is good occlusion and balance of the masticatory muscles, the act of chewing improves cranial function. Why was it that when Silverman⁴⁸ noted poor occlusion upon awakening in the morning as a result of the stresses on the skull from the sleeping position, normal occlusion always returned after eating breakfast if not before? When the muscles function in a balanced manner and the occlusion is correct, contraction of the masticatory muscles repeatedly activates the skull by the same lever forces described in maximum jaw opening. With this mechanism the body has a self-correcting approach to return normal function after a blow on the head or the more gentle stresses that occur with everyday life. A student's cranium can be disturbed when he sits at a desk with his arm and hand propped under the zygomatic bone to hold his head. A welder's helmet or other headgear can sometimes disturb the cranial mechanism, but normal function returns with a yawn or chewing. Even walking, with the alternate contraction of the sternocleidomastoid and upper trapezius, can re-activate normal function in the skull. It is fortunate that all these autocorrection mechanisms are present, or normal health would suffer.

Unfortunately, balanced muscle action and occlu-

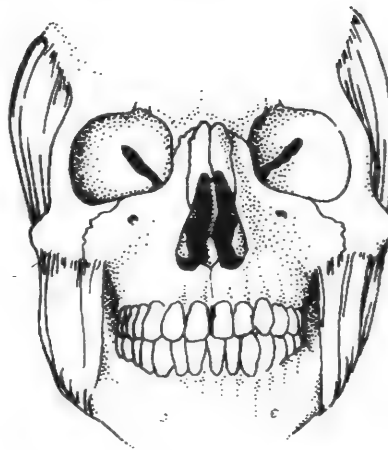
sion are not always present. When they are not, chewing becomes a detrimental factor rather than an asset. When there is poor occlusion, the mandibular elevators pull into the cranium in an imbalanced manner. Apparently it is the repetition of this mechanical stress into the cranium which causes disturbance. If an individual bites hard on a resistant object between the molars on only one side of the jaws there is obviously an imbalance present, but it is only transitory; continued chewing in a more balanced manner often returns normality to the skull. It is continued imbalance of malocclusion which creates problems.

The imbalanced platform from which the muscles work is not the only problem when there is malocclusion; there is considerable evidence^{5, 7, 37, 39} that malocclusion causes several changes in the action of the masticatory muscles. The muscles, as observed on electromyography, may be firing stronger on one side than on the opposite. There may be improper timing of the muscles in the open-close-clench cycle, and there may be disturbance in the jaw-opening reflex. The cranial primary respiratory mechanism can be corrected when these patterns are present, but as soon as an individual bites down or chews, the cranial disturbance returns. This type

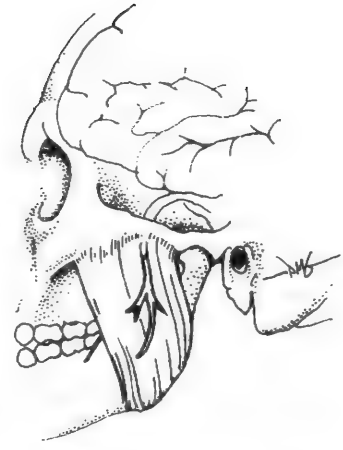
Cranial and Stomatognathic Area Interdependence



10—1. Malocclusion causes muscle force to distort the cranium, creating or perpetuating cranial faults.



10—2. Local muscle imbalance can pull on the cranium in an imbalanced manner.



10—3. Cranial faults with resultant entrapment of cranial nerve V can cause muscle imbalance to perpetuate the cranial faults.

of recidivism is not always due to malocclusion. The muscle itself can be dysfunctioning as a result of improper neuromuscular spindle cell activity, or improper signaling from a periodontal ligament receptor could be creating the problem.

Further interaction of body function or malfunction is observed; regardless of whether the problem begins with a malocclusion or with cranial faults, the two interact with each other and compound the problem. Cranial faults can cause neurologic disturbance in the muscles of mastication by peripheral nerve entrapment of cranial V. The muscles of mastication can be disturbed just as other muscles in the body. The neuromuscular spindle cell or Golgi tendon organ may be traumatized by overcontraction or overstretching of the muscle. The fascia and muscle may not be in harmony, or trigger points may be present. These are local problems within the muscle itself. The cause of the muscle's dysfunction could be remote, such as poor lymphatic drainage, blood vascular supply, peripheral nerve entrapment, or imbalance in the meridian system. Local causes of muscle dysfunction are usually from overstretching or overcontracting the muscle. This may happen as a result of prolonged dental procedures, stretching exceptionally wide to bite on an apple, or biting very hard on some object. It appears that the muscles are more susceptible to local trauma when nutritional deficiencies are present. Any of these problems may cause the muscle to be weak or hypertonic, throwing the integration of the masticatory muscles out of balance.

The muscles of mastication do not lend themselves well to direct muscle tests. Indirect testing procedures have been developed in applied kinesiol-

ogy to determine if the timing and action of the muscles are correct. Evaluation appears to be of the proprioceptors and their integration, along with interpretation and organization of nervous system control. Imbalance of only one muscle can result in a prematurity in an otherwise normal occlusion. The prematurity stimulates the periodontal ligament proprioceptors, ultimately changing the jaw-closing engram to attempt improved occlusion. But if the muscle cannot react correctly because of its own inability to function normally, it cannot respond to the new engram; thus confusion continues within the system. If the imbalance of the masticatory muscles persists for several days or longer, the teeth will start to shift from the constant pressure.²⁹ Now even if the cranial dysfunction can be corrected by the body's own mechanisms, or if a physician makes the correction, there is a malocclusion which may need treatment.

A vicious circle of interaction may develop from an initial disturbance of the proprioceptors in the periodontal ligament, as indicated by the neurologic tooth condition in applied kinesiology evaluation. This problem could develop from biting on a piece of bone while eating, which severely rocks or twists a tooth. Microtrauma in the periodontal space may cause the proprioceptors to send afferent impulses not in keeping with the actual status of the tooth. If this persists, the engram regulating mandibular closure is altered to fit the aberrant impulses. In essence, the system is interpreting a prematurity or some other noxious factor when it is not actually there. The resultant muscular imbalance may cause a cranial disturbance, throwing structure off-balance as a result of entrapment of the cranial nerves. Now

there is a frank problem of a potentially wide magnitude; the original problem was limited only to the disturbance of the tooth proprioceptor, which in time might have repaired itself. The muscles of mastication can become more involved as a result of cranial V disturbance, and occlusion and temporomandibular joint activity can be further disturbed; the vicious cycle has developed. Many examples can be

given showing how malfunction in a very limited area can start an entire cycle of wide-ranging effects. The discussion here is limited to the stomatognathic area, but it does not end there. It is possible for disturbed function to develop in just about any area of the body from a simple initiating factor of a neurologic tooth as described.

Swallowing

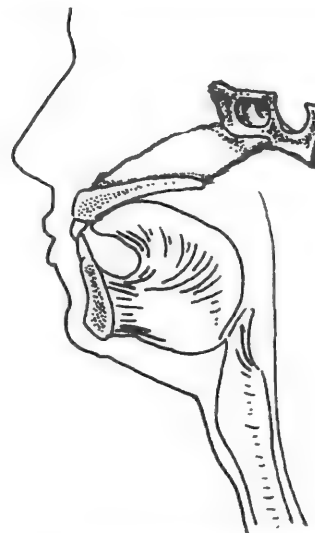
Each individual develops a swallowing pattern which is an engram consistent for that person. Although there is a specific repeatable pattern in individuals, it is not consistent between individuals.²⁴ The pattern of swallowing can either enhance stomatognathic function or be detrimental to it.^{15, 16, 19, 42} It is the repetitive forces on the stomatognathic system that become important. A person swallows from 1500-2000 times every twenty-four hours. Some believe this averages approximately two times per minute while awake and once per minute while asleep.^{2, 19, 42} The importance of correct swallowing is emphasized even more by the amount of force exerted throughout a day by swallowing. Measurements have shown that if chewing forces are calculated for the whole day, they give rise to only half as much force as swallowing.²⁸

In normal swallowing, the tip of the tongue presses against the palate just behind the central incisor teeth. The middle of the tongue forces against the hard palate, and the posterior part is tipped at a 45° angle against the pharyngeal wall. The mandibular elevators contract to stabilize the mandible for subsequent suprahyoid muscle activity. The teeth are in complete intercuspation. The lips are sealed, the middle of the tongue rises to the palate, and the swallowing pattern is accomplished with negative intraoral pressure. As the tongue is activated from anterior to posterior, the food bolus is dumped with little or no anterior or lateral pressure against the dental arches. Along with tongue activity, the hyoid bone is elevated by the suprahyoid muscles, which are the geniohyoid, mylohyoid, digastric, and stylohyoid. The bolus passes through the oropharyngeal isthmus into the oropharynx. This first stage of swallowing is either voluntary or involuntary; from this point the mechanism is all reflex activity.

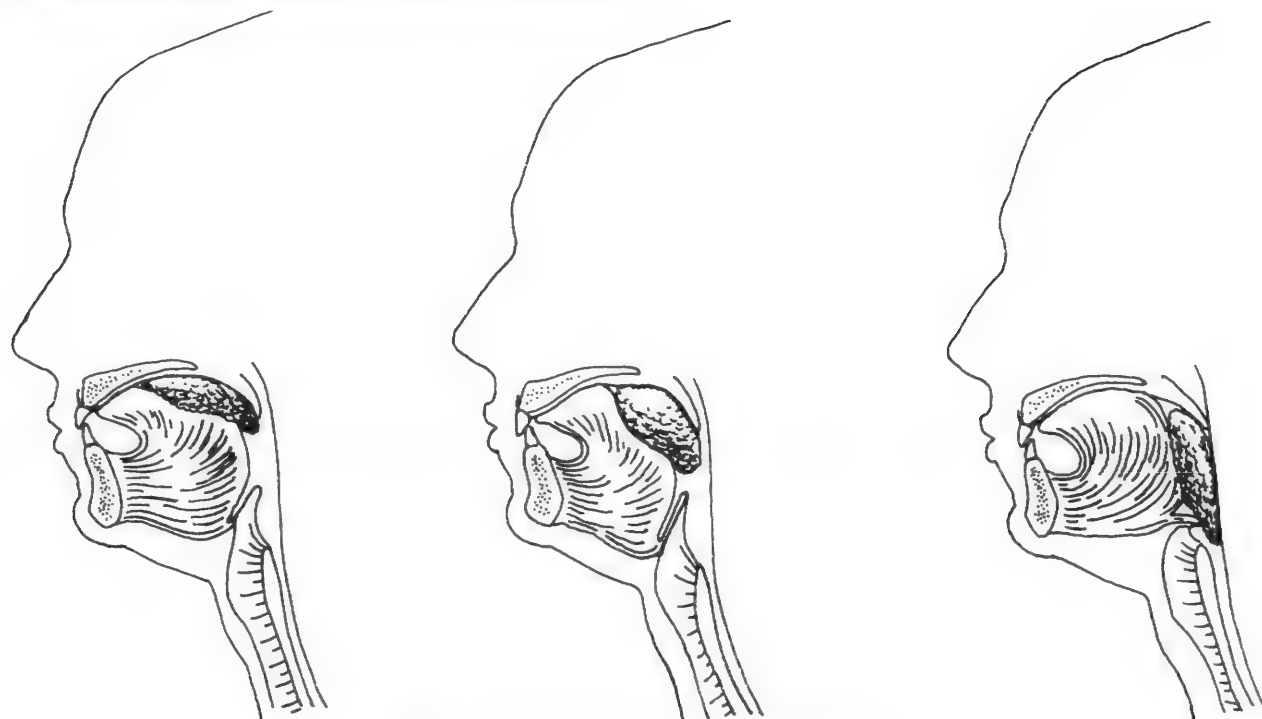
The position of the tongue to the palate has been confirmed by cinefluorographic sequences of deglutition.²⁵ The force exerted is 52 gm per square cm against the anterior area of the palate with spontaneous swallowing. With voluntary swallowing of saliva, force is greater in the amount of 112 gm per square cm.²⁷

Analysis of the forces present in a normal swallowing pattern allows us to understand why Silverman⁴⁸ was able to more rapidly return his occlusion to normal after the skull had been distorted from an external force. As the tip of the tongue presses against the anterior palate, a force is transmitted through the vomer to the rostrum of the sphenoid to slightly mobilize the sphenoid. At this point another factor must be considered. In the normal swallowing pattern forces are balanced; normal strength of the lips counteracts and minimizes the activity of the tongue at the anterior palate. At about the same time the tongue initiates its action, the mandibular elevators contract to bring the teeth into centric occlusion, locking the mandible to provide a stable base from which the suprahyoid muscles pull. Thus in every swallow, the teeth are brought into centric occlusion with a moderate amount of force.

With every swallow the same forces which have been discussed during the act of mastication are directed to the cranium. If the structures are all in harmony and muscles are functioning in a balanced manner, swallowing enhances cranial motion; on the



10—4. Tip of tongue at incisive papilla.



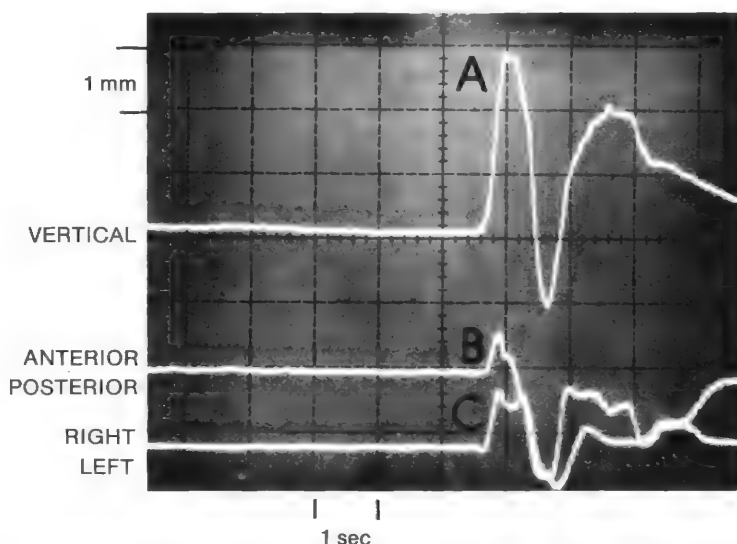
10—5. Progressive stages of swallowing.

other hand, if there is any type of imbalance in the total mechanism, the cranial primary respiratory mechanism will be adversely influenced. If a prematurity is present, there may be even greater disturbance during swallowing than during chewing. It has been shown that during the act of mastication there is no actual tooth contact because food remains between the teeth and the stroke ends short of tooth contact.²⁵ The bolus between the teeth could cushion a slight prematurity to lessen the impact of its existence. During swallowing there is direct tooth-to-tooth contact with activity of the mandibular elevators.

When the swallowing pattern is improper, smooth integrated motion fails. Ravins⁴² recommends a method of evaluating swallowing "... by placing a hand on the throat and feeling the movement of the hyoid bone as the patient swallows." In coordinated swallowing the hyoid moves in a smooth circular pattern. The abnormal pattern loses the smooth action, delays, and becomes jerky, with an elliptical movement; this is associated with tooth apart swallowing.⁴⁹

When occlusion and the muscular

activity of the swallowing mechanism are balanced, the forces placed into the skull enhance cranial primary respiratory function. It is when any one — or many — of the factors of the swallowing pattern deviate as to timing or direction that disturbance is caused, not only to the cranial primary respiratory mechanism, but possibly to the occlusion as well.



10—6. Mandibular Kinesiograph tracing (see page 319) of mandibular movement during swallowing of an individual with malocclusion. The straight lines on the left represent the stable position of the mandible when at rest. Point A represents almost 3 mm of closing to intercuspatation. Point B indicates .05 mm of anterior slide, and point C almost 1 mm of right slide.

Head-To-Shoulder Girdle Musculature

The muscles connecting the head to the shoulder girdle are not typically thought of as a group for study. Usually the muscles are grouped as postural muscles, deep spinal muscles, supra- and infrahyoid muscles, and muscles of mastication. Here we are going to consider them first as individual groups with the complex activity of each, and then put the total muscular pattern with interactions into perspective. In the final analysis there is no separate action taking place in this area; it is a closed kinematic chain with each so-called division capable of affecting the total complex, or the total complex capable of influencing one separate area.

Complexity develops as a result of the numerous responsibilities of this area for normal body function. The muscles are responsible for head balance, which is very important to equilibrium and orientation in space. Muscles that contribute to this closed kinematic chain are responsible for mastication from one group and swallowing from another, but as we have seen and will continue to study, the muscle groups of mastication and hyoid are interrelated in these activities.

For study purposes this discussion is primarily limited to the muscles in the closed kinematic chain of the head-to-shoulder girdle. Of course, interaction is not limited to this area, and ultimately in our study of applied kinesiology, muscles, structures, and functions remote from the current discussion are integrated. For now it is adequate to recognize the influence of the lower body on this area. For example, there is rhythmic, alternate facilitation and inhibition of the sternocleidomastoid and upper trapezius muscles when an individual walks or runs. These muscles pull into the cranium, influencing the primary respiratory system. In addition, other activities such as extensor muscle facilitation from the positive support mechanism of the proprioceptors of the foot, as well as interactions of the cloacal and gait reflexes, influence posture and balance. These factors must be kept in mind because any structural strain may disturb normal activity of the stomatognathic system. As this discussion focuses on the muscles of the head-to-shoulder girdle in their usual grouping for study, keep in mind the interactions of the total complex which will be presented later.

POSTURAL MUSCLES

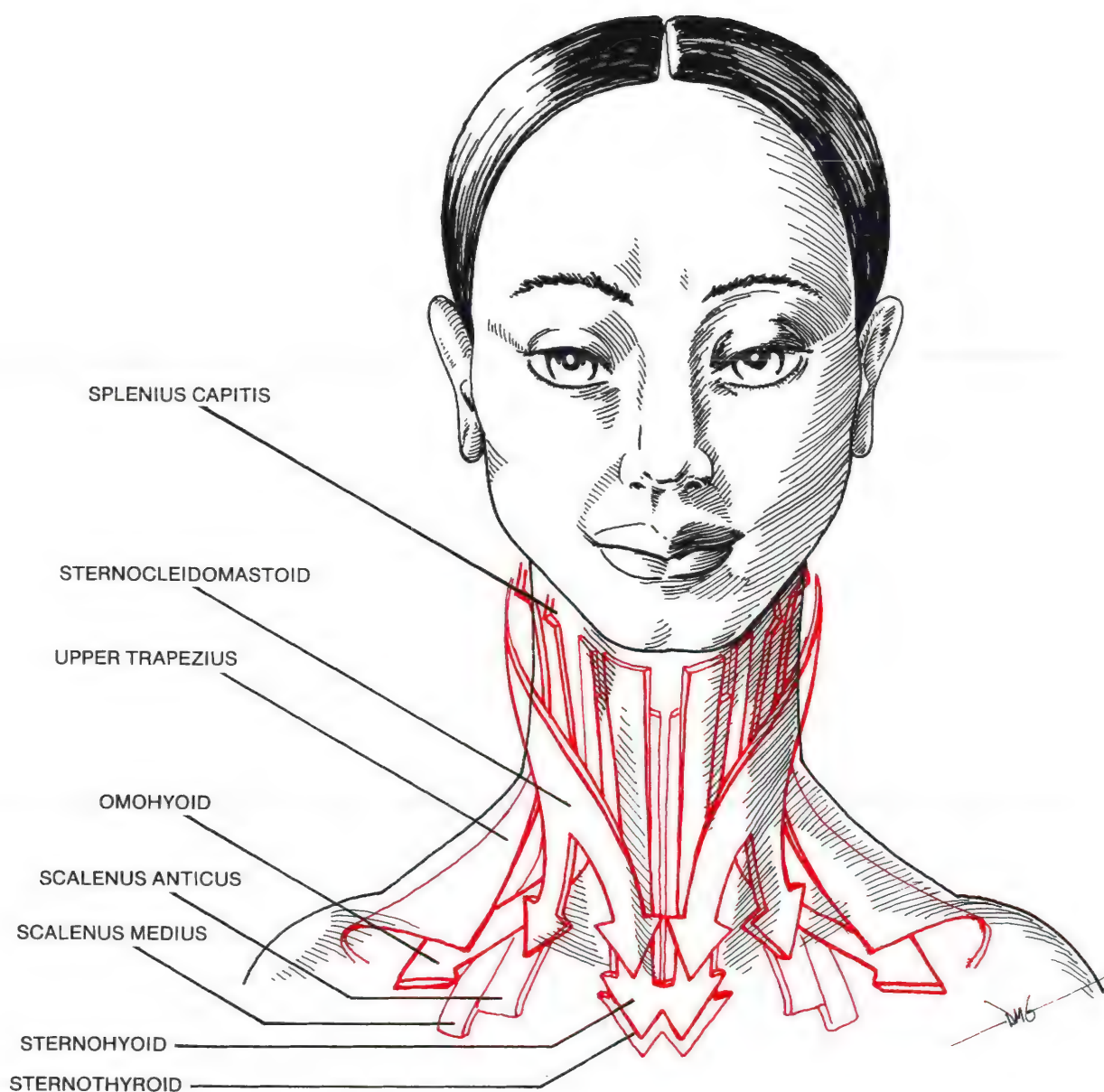
The primary postural muscles of this area are the sternocleidomastoid and upper trapezius, with some contribution from the levator scapula. These muscles have the major responsibility for head leveling. If the muscles had no other responsibility than this, their importance to total body organization would be paramount. The importance of head leveling is emphasized by people like Alexander,¹ Barlow,³ Tinbergen,⁵³ and Dart.^{8, 9, 10} They relate how important it is to have habitually correct head carriage. Improper head position may be a result of poor postural habits learned early in life, or it may develop as a result of injury or habits later in life.

The head and neck can be out of balance as viewed from anterior to posterior, thus comparing the right with the left side. In this instance we are dealing with basically equal muscles, as one side is the mirror image of the other. Balance can also be viewed laterally, where the anterior muscles in no way parallel the posterior.

A very interesting presentation on body organization is tendered by Dart.^{8, 9, 10} He states that the principles set forth "... by Alexander (are) appropriate because (they are) based on the fundamental

biological fact that the relation of the head to the neck is the primary relationship to be established in all proper positioning and movement of the body." He presents convincing evidence of the importance of this relationship. When the body is not correctly oriented to the head, the neck righting and body-on-head reflexes¹⁸ are directly influenced. Most physicians are not concerned when they observe that a patient consistently holds his head in an abnormal position; in fact, very often it is not even noticed. Is it important?

When neck righting and body-on-head reflexes are stimulated, compensatory reactions take place throughout the body in an attempt to orient with the head position. This can be summed up with the statement, "As the head goes, so goes the body." The body is constantly trying to orient with the head position. This is problematic enough in a static standing or sitting posture; it becomes even more involved when an individual walks or runs. Disorganized facilitation and inhibition of the upper trapezius and sternocleidomastoid muscles can pull into the cranium in an imbalanced manner, causing lack of harmony between the cranial and sacral



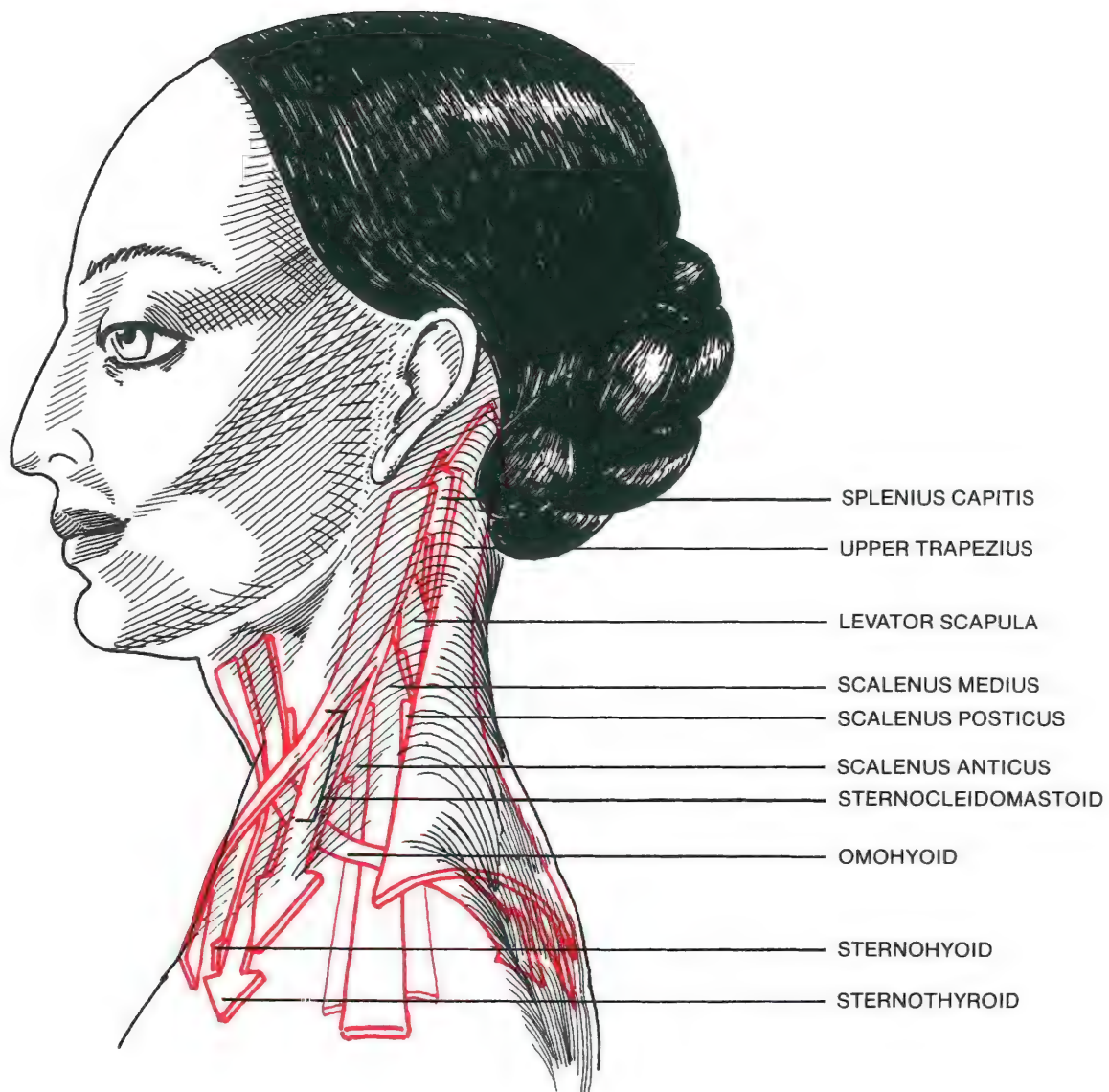
10—7. Symmetrical muscle balance from the right to the left side.

primary respiratory systems. This can cause or perpetuate cranial faults.

Imbalance of the sternocleidomastoid and upper trapezius muscles may be a direct result of cranial faults since both of these muscles receive part of their innervation from cranial nerve XI. Whenever there is an imbalance of the sternocleidomastoid and upper trapezius, very close attention should be given because of the unique supply of these muscles; they receive both cranial and spinal innervation (see page 133).

There may be a localized disturbance in the muscle(s) from improperly functioning muscle proprioceptors, trigger points, or disharmony of the muscle with its fascia. In this case, treatment to the proprioceptors, spray and stretch technique, or fascial release treatment is needed to return the muscle(s) to normal. With the muscles now functioning normally, the head and neck are balanced and structure returns to normal. This is an example of a primary problem in the head-leveling muscles.

If the imbalance is not caused by a primary



10—8. Balance of the head-on-neck, and neck-on-body, is from regulation of muscles which are much different when compared anterior to posterior.

dysfunction of these postural muscles, efforts to level the head by working with the muscles directly, changing habit patterns, or exercise, will be unrewarded. The problem may be in some other area of the closed kinematic chain, or remote in the body. As usual, the answer is to find the primary condition and effectively treat it.

There are additional muscles in the cervical region such as the scalene, longus, splenius, and semispinalis groups, as well as the smaller intrinsic

muscles of the cervical-occipital region. These muscles are involved primarily with subluxations of the cervical spine, including the occipital bone. Either subluxations or muscle imbalance can influence the neck righting and body-on-head reflexes; each is somewhat involved in the closed kinematic chain. Similar attention in evaluation and, if necessary, treatment should be given to these muscles as has been indicated for the sternocleidomastoid and the upper trapezius.

MUSCLES OF MASTICATION

The importance of the masticatory muscles to the cranium has already been delineated. This includes the necessity of balanced centric occlusion and normal function of the periodontal ligament receptors. These are important not only in the act of chewing and other usually thought of mandibular movements; they are probably more important because of frequent tooth contact and contraction of the mandibular elevators throughout the day and night during swallowing. Swallowing requires elevation of the hyoid, accomplished by the suprahyoid muscles. Since the mylohyoid, geniohyoid, and anterior belly of the digastric all originate on the mandible, it must be stable for these muscles to function. If the hyoid is to be elevated by these muscles, they must pull from a solid base. The only way for the mandible to provide this base is by contraction of the mandibular elevator muscles. This happens on an average of every half-minute during

the daytime, and at night every minute. Disturbance in the mandibular elevator muscles or a proprioceptor of the periodontal ligament would thus send improper information into a highly integrated and important area of the nervous system.

Consideration of imbalance of the masticatory muscles is very important because of the frequency of disturbance in them. Weinberg⁵⁶ lists as precipitating factors of muscle imbalance sudden stretch, acute muscle fatigue, and acute stress syndrome. These factors usually relate to a previous muscular stress originating in any portion of the stomatognathic system.

An imbalance of the muscles of mastication, whether it be in deviant opening or closing of the mandible or other mandibular malposition, will influence hyoid muscle activity, thus changing the afferent information from their neuromuscular spindle cells.

HYOID MUSCLES

The hyoid bone is suspended by muscles much like a hammock; it has no bony communication. Its function is to provide an attachment point for muscles. Their activity in mastication, mandibular movement, swallowing, and phonation is not well-known and is only briefly described in the literature. Are these activities the only contribution this neuromuscular complex makes to body organization? Goodheart¹⁷ has compared hyoid suspension with a gyroscope in a guidance system. A gyroscope is flexibly mounted in an object such as a missile, ship, or airplane so that it maintains its equilibrium. Sensors relay information about any change of position between the vehicle and the gyroscope, providing a type of feedback. This information is used by an automatic guidance system, such as an autopilot, or by a pilot to know the airplane's orientation in space. If the airplane and the gyroscope are not level with each other, the instrument will show this disparagement.

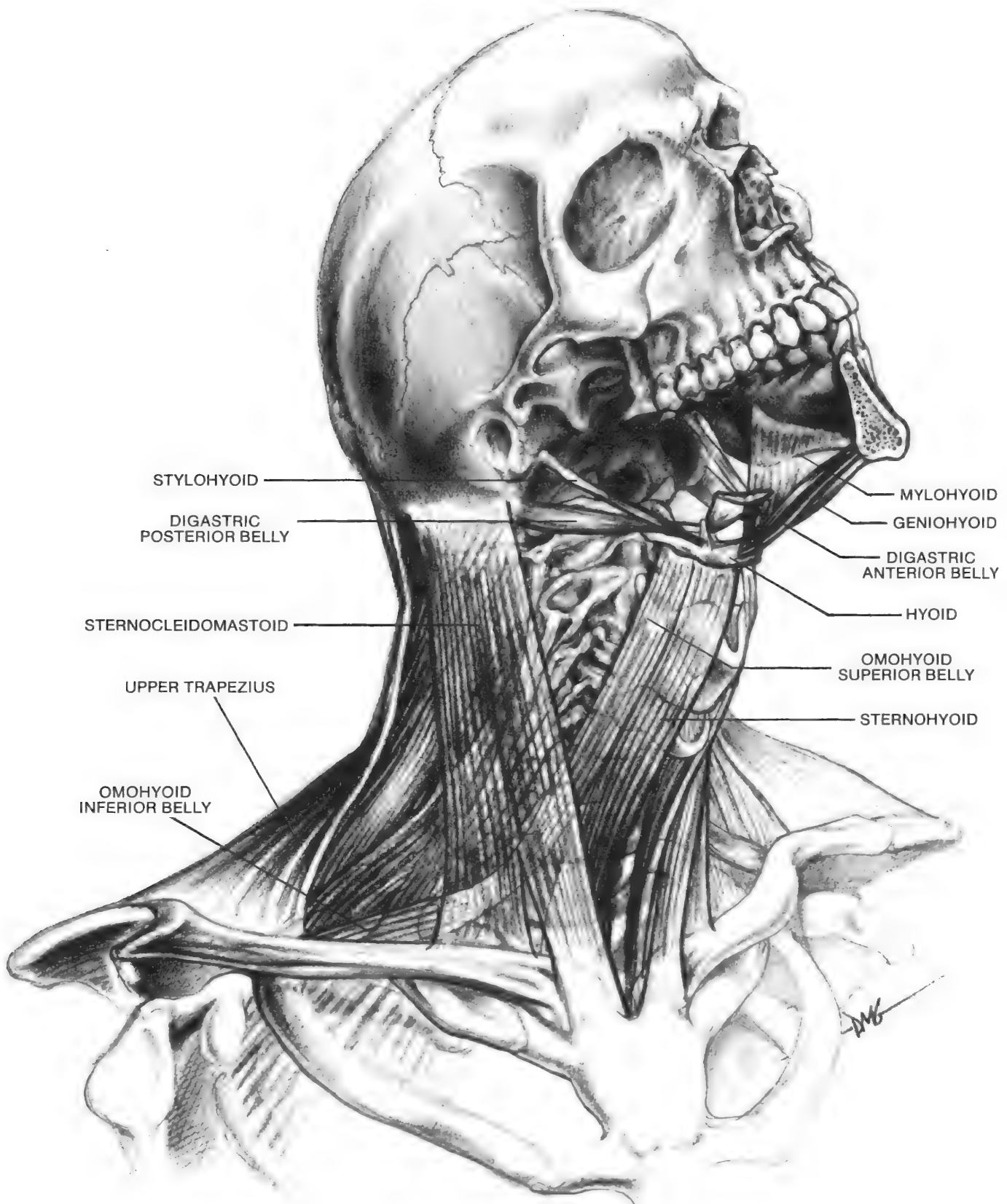
There are several examination procedures in applied kinesiology which indicate that the neuromuscular system of the hyoid provides analagous information for body equilibrium. The proprioceptors of the hyoid muscles appear to provide afferent information which is compared with total body position or orientation in space. (This is discussed more thoroughly in Chapter 13 on the hyoid.) The suprahyoid muscles have their origins on the skull and the mandible, while the infrahyoid muscles originate from the sternum, clavicle, scapula, and thyroid



10—9. Gyroscopic instrument indicating airplane's orientation in space.

cartilage. With the muscles originating from such varying locations, any change of the head-neck-shoulder girdle relation is going to cause transmission of considerable information from the neuromuscular spindle cells of the various muscles. The central nervous system has complete information as to the exact location of the hyoid bone at any given time.

The stylohyoid and the posterior belly of the



10-10.

Cranial and Stomatognathic Area Interdependence

digastric originate on the cranium, giving specific head-to-neck information. An imbalance of these muscles may influence the cranial primary respiratory mechanism by an imbalanced pull on the structure. The sternohyoid, originating from the sternum and clavicle, relates the neck with the shoulder girdle.

The omohyoid, originating clear down at the scapula, orients that structure with the neck. These origins also cause the hyoid to be part of the anterior portion of the closed kinematic chain of the head-on-shoulder girdle complex.

MUSCLES OF HEAD-ON-SHOULDER GIRDLE CORRELATION

All the muscles discussed make up a closed kinematic chain, with each muscle interdependent on the activity of the other muscles.^{4, 6, 14, 26, 38, 51} The closed kinematic chain is beautifully illustrated by a diagram originally presented in 1949 through the University of Illinois⁵⁴ (Fig. 10—11). It is amazing that more interest and attention has not been given to improving the balance of these structures and muscles in this period of time.

The activity of the posterior cervical group (A-B) must equal the activity of the anterior cervical group (C-H) for equilibrium to be present. Inhibition of the posterior group with no change in the anterior group will cause the head to tilt forward. Increased activity of the posterior group with no change in the anterior group will cause the head to tilt backward. Change in any section of the anterior group, whether it be the mandibular elevators (C-D), suprahyoid (E-F), or infrahyoid (G-H), will influence the entire group. To maintain the head in equilibrium and have mandibular elevation, there must be contraction of the mandibular elevators concurrent with inhibition of the suprahyoid muscles; otherwise, the head would bob up and down when a person chews or talks because the anterior portion of the closed kinematic chain fails to equalize the posterior division.⁵⁷

Postural change influences the activity of the muscles of mastication³⁰ and also the rest position of the mandible.¹² Even more important is the interaction of masticatory muscles with head position related to the occlusion. Funakoshi et al.¹⁴ demonstrated balanced electromyographic responses in the muscles of mastication with different head positions when the occlusion was normal. In the presence of a malocclusion artificially caused by an overlay set on a molar to cause a premature contact, the balanced response became unbalanced. In the case of malocclusion which was equilibrated to correct occlusion, the electrical activity of the muscles changed from imbalanced to balanced.

Goodheart often points out the accuracy of Alexander's^{1,3} observation of how inefficiently people use their bodies by how they react to the request to open their mouths. Rather than dropping the mandible, which is the effective and functional way to

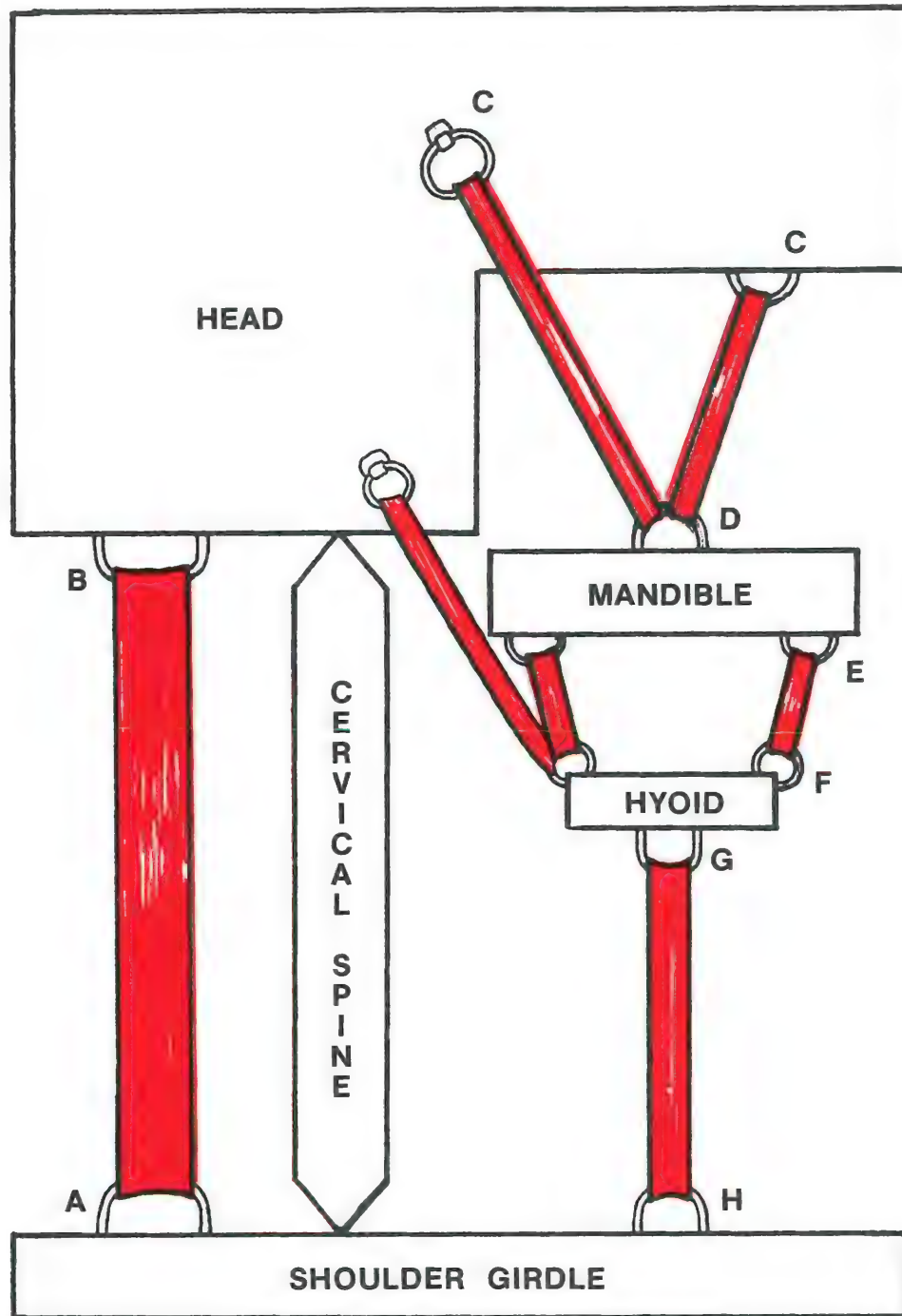
open the mouth, people tilt their heads back and, as Goodheart says, they "... open their heads." As we can see from the diagram, the hyoid and muscles of mastication complex provide considerable anterior support to the closed kinematic chain. Very dynamic activity takes place in the process of chewing and swallowing. During mastication the mandibular elevators are activated with great complexity to produce the grinding action of chewing. As the jaw closes there must be inhibition of the suprahyoid muscles to maintain hyoid stability. While the suprahyoid muscles relax, the infrahyoid muscles stabilize the hyoid's position. The next activity is to swallow. The hyoglossus must relax to allow the tip and middle of the tongue to elevate and press against the palate. The suprahyoid muscles contract, elevating the hyoid; this is allowed by the infrahyoid muscles.

Allow one single improper volley of nerve impulses into this complex picture, and we can observe the entire mechanism going haywire. It does not matter whether the improper impulses come from a periodontal ligament receptor, a neuromuscular spindle cell in a muscle of mastication, the hyoid group, muscles of the tongue, or any one of the other sources. A weakening or strengthening of the muscles of mastication-hyoid complex changes the balancing requirements from the posterior cervical group. A change of one anterior group influences the other groups. If there is an imbalance from right to left, the head-leveling mechanism is thrown off-balance and the equilibrium proprioceptors are stimulated. This causes muscle pull into the cranium in an imbalanced manner. Cranial faults may develop, producing cranial nerve dysfunction. As a result of cranial nerve dysfunction the muscles of mastication, hyoid, sternocleidomastoid, and upper trapezius may be improperly innervated. The stomatognathic system can be further adversely influenced by facial muscles improperly contracting, causing additional disturbance in the cranial primary respiratory mechanism. Cranial faults may also cause the eyes to function improperly, affecting the visual righting reflex. The labyrinthine reflex, innervated by cranial nerve VIII, can be affected in a similar manner. Disturbance in these equilibrium proprioceptors may

cause further imbalance of head leveling and severe confusion within the nervous system.

How far can we go with this hypothetical example? It is amazing what long-range effects a seemingly small problem can create. Mrs. Jones'

neck pain or headaches may result from something as simple as biting on a chip of bone in a salisbury steak a week ago. This may cause disturbance of the proprioceptors in the periodontal ligament — which fail to return themselves to normal — disturbing the



10—11. Block diagram of the closed kinematic chain of the stomatognathic system. The sternocleidomastoid muscle has been left out for clarity. Redrawn and modified from the University of Illinois Telephone Extension Program, Current Advances in Dentistry, 1949.

Cranial and Stomatognathic Area Interdependence

entire closed kinematic chain of the head-on-neck muscles. But the problem could become far-reaching. If cranial faults develop and the vagus nerve becomes involved, digestive disturbances may result. The physician and patient may have no idea how the problem began. Everything in the body happens for a reason; it is the physician's challenge to find that basic underlying cause.

All this may seem highly complex and formidable to examine and evaluate. Certainly the interactions

that take place are complex, but with a systematic method of evaluation, the primary imbalance which initiates the multitude of disturbances can readily be found. This will become obvious as we proceed with the interactions, examination, and treatment of the individual systems. For now it is important to understand the magnitude of these interactions and the fact that the symptomatic area may not be the primary factor needing evaluation and treatment.

Development of Neurologic Disorganization

Disturbances developing within the stomatognathic system follow the same basic pattern in which neurologic disorganization develops in any other portion of the body, as described in Volume I. During examination of a symptom, abnormal findings may seem random. The patient's complaint may well be caused by muscular hypertonicity. Treatment directed to the muscle may provide relief, but care must be taken to determine whether it is actually a primary involvement. If not, the treatment — even though it may relieve the symptoms — will only be temporary, or another problem will manifest itself in some other area, again secondary to the real undiscovered dysfunction. It is all too common for the presenting symptoms about which a patient complains to be secondary in nature. Because this is the individual's complaint, the physician may concentrate his efforts in this area and miss the remote primary cause of the disturbance. This interaction is exemplified as strongly in the stomatognathic system as anywhere in the body.

The reflex arc is the simplest example of how something can go awry, causing a symptom remote from the actual disturbance. In the reflex arc a receptor is stimulated. The afferent nerve transfers information to the cord, the efferent system is stimulated, and a reaction occurs as a result of the initial receptor stimulation. If all is working properly, the reaction meets the needs of the body as presented by the initial stimulation of the receptor. If for some reason stimulation of the receptor was improper, it is obvious that the message and the body's reaction will not be in keeping with its needs. This is an illustration of a segmental reflex. The mechanism gets more complicated if the reflex is propriospinal or suprasegmental. The stimulation and following reaction can be within structural components (somatosomatic reflex), from structure to organs (somatovisceral reflex), or from organs to organs (viscerovisceral reflex).^{45, 55}

To put into perspective the secondary, remote,

and possibly symptomatic disturbances that develop, it is necessary to understand how the nervous system picks up information to be processed within its various reflexes and by higher centers. The origin of the activity is with the receptors, which have standard classifications²¹ as mechanoreceptors, thermoreceptors, nociceptors, electromagnetic receptors, and chemoreceptors. In this discussion we will add mental receptors.

Mechanoreceptors

Mechanoreceptors of many types are extensive throughout the body. Proprioceptors are a type dealt with extensively in applied kinesiology. Proprioceptors can be divided into those located in the muscles, joint and skin, and those for equilibrium.^{18, 55} When muscle proprioceptors send improper information, it appears they do so as a result of trauma. This may be swelling within the neuromuscular spindle cell, causing improper stimulation, or possibly adhesions may ultimately develop in the delicate structure. The trauma may result from overstretching or overcontracting of the muscle, or direct trauma to the receptor.

Joint receptors signal articular movement of as little as 2°. From this information, engrams are developed in the nervous system which organize muscular activity for the control of this articulation; in addition, they relay information to the rest of the body about this joint's activity and position. A joint subluxation causes stimulation of the proprioceptors which is not in keeping with the developed engrams. The proprioceptors in the periodontal ligament are excellent examples of joint receptors; they can be adversely influenced by trauma to a tooth and create a plethora of disturbances until returned to normal by the body's own repair mechanism or by the therapeutic endeavors of a physician.

Much of the body's structural organization comes from the equilibrium proprioceptors. These are classified differently by various authorities. Often the labyrinthine, visual righting, neck righting, and

body-on-head reflexes are considered in this group. A much broader grouping could be made since many of the mechanoreceptors in the body deal with equilibrium, including some of those in the feet, pelvis, spine, and other areas. In the final analysis, probably all mechanoreceptors in the skin, joints, muscles, etc., contribute some to the process of equilibrium.

The integration of the tonic neck reflexes with jaw muscles was demonstrated by Funakoshi and Amano.¹³ Flexion of the head on the neck inhibited electrical activity of the bilateral masseter, temporalis, and digastric muscles. The influence was abolished by cutting the first three cervical nerves, indicating the tonic neck reflex involvement.

Also included in the mechanoreceptors are those in the cochlea for hearing, and baroreceptors generally in the carotid sinuses and aorta. There appear to be baroreceptors in the lymphatic system as indicated by the retrograde lymphatic evaluation in applied kinesiology.⁵⁵

Thermoreceptors

It is unclear how much influence the thermoreceptors have on body function in the context of our discussion. It is known that there are certain types of imbalance in the meridian system which cause predictable areas to be hypo- or hyperthermic. Whether this can stimulate a somatovisceral reflex to adversely influence circulation is unclear. As more data becomes available, it may reveal that remote actions not in keeping with body harmony can develop as a result of vicarious stimulation of these receptors.

Nociceptors

Pain sensation enters the nervous system via nociceptors. Reaction is generally to protect the body. An example of how this normal activity may cause disturbance as a result of the natural protective mechanism is seen in the stomatognathic system as a prematurity. As Dawson¹¹ and others point out, an engram will develop to avoid contacting a painful prematurity. This muscular activity to "improve the

occlusion" establishes an imbalanced muscular pull into the cranium and may adversely affect it while trying to adapt to the prematurity.

Electromagnetic Receptors

The rods and cones of the eye are those generally listed as the electromagnetic receptors.²¹ Applied kinesiology recognizes points on the meridians of the body (acupuncture points) as electromagnetic receptors. Evidence for this is presented in Volume III of this series. It appears that considerable input is given to the nervous system by these receptors. Much study is needed in this area, which is a new field of investigation for the Western world. It seems apparent that the body's electromagnetic patterns and other energy fields have an important influence on the nervous system.

Chemoreceptors

The status of the body's chemical environment is continuously monitored by the nervous system from information provided by the chemoreceptors. Possibly some of the most important chemoreceptors are in the hypothalamus. Again, this is an area where the data base of current knowledge is severely limited. There is a dearth of current knowledge about how the body is immediately influenced by various chewed chemical substances, which may include nutritional or harmful chemicals. It is important to recognize that the nervous system is significantly influenced by chemical receptors. This is thoroughly discussed in its relationship to applied kinesiology in Volume V of this series.

Mental Receptors

It is well-known that a person's mental attitude and various mental processes influence health in either a positive or a negative way. Mental receptors are not generally included in a classification of the nerve receptors of the body. As we continue our discussion of primary and secondary disturbances, these receptors, as well as the others, must be kept in mind.

MODEL OF PRIMARY AND SECONDARY CONDITIONS

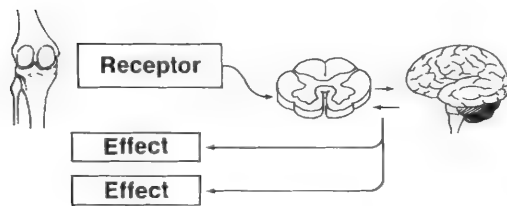
This model is based on the principle that for every action there is a reaction. The action begins with the stimulation of a nerve receptor; we follow the reaction that takes place as a result of that neurologic stimulation. In this example, the joint proprioceptors of the knee are stimulated as the joint moves. The information is transmitted to the central

nervous system over the afferent pathways. Some reflexes occur at the intersegmental level, some at the propriospinal level, and possibly some at the supraspinal level. As a result of this action, various neurons in the efferent system are stimulated to create both facilitation and inhibition of muscular activity. This is normal activity in keeping with the

Cranial and Stomatognathic Area Interdependence

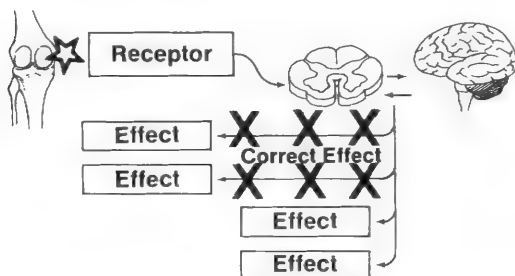
movement at the knee.

This example relates to activity throughout the body. In place of a receptor at the knee, we could just as easily use a mechanoreceptor in another area of the body, such as the temporomandibular joint, muscles of mastication, periodontal ligament proprioceptors, joints of the cervical spine, etc. Other types of receptors could also be involved, such as chemoreceptors, nociceptors, baroreceptors, etc. The point of the illustration is that when a receptor is stimulated the central nervous system reacts on that information, causing various effects throughout the body.



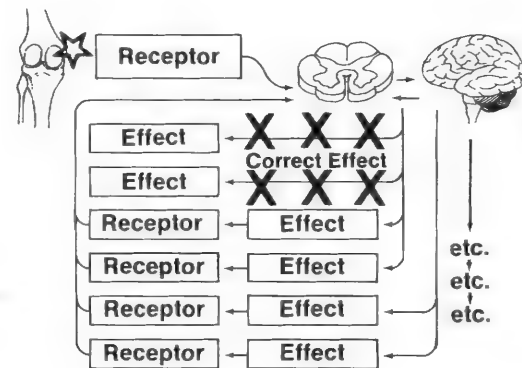
10—12. Normal receptor — effect.

Unfortunately, the stimulation of receptors is not always appropriate for the body's needs. An example of this is trauma to the knee which, although slight, causes a subluxation which stimulates the joint proprioceptors in a manner not in keeping with the knee's activity. As the knee goes through its range of motion, the joint's abnormal activity stimulates the joint proprioceptor in a different manner than before the injury. The information picked up by the afferent system is processed in a manner similar to the normal management discussed above; however, it is now processing improper information from the disturbed joint receptors. Again, the efferent system is responsible for inhibition and facilitation of specific muscles, which are not now in keeping with the actual joint activity. Here again there is action and reaction, but it is based on improper information from the afferent system.



10—13. Reaction to improper stimulation of the receptors is incorrect effects.

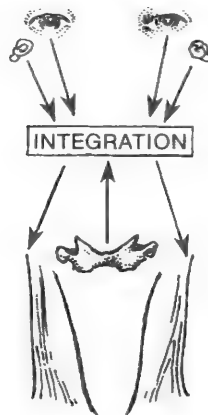
The new but inappropriate effects stimulate additional receptors which are processed by the central nervous system, and produce new effects not in keeping with the needs of the body. The new effects may be remote from the knee and very much in keeping with what the body thinks is taking place. For example, the proprioceptors of the knee contribute some information about the gait mechanism to the central nervous system. When an individual walks there is facilitation and inhibition of the sternocleidomastoid and upper trapezius, as well as many other postural muscles. If facilitation and inhibition of these muscles are improper, hypertonicity and consequent pain may develop. The patient with the condition described may have neck pain or suboccipital headaches as a symptom.



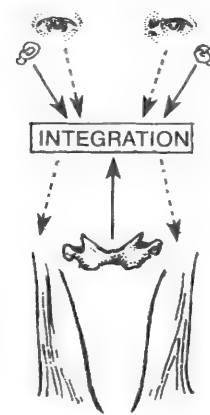
10—14. This disorganization can go on and on.

This chain of events can continue indefinitely. The imbalance of the sternocleidomastoid and upper trapezius also stimulates receptors. Again, the central nervous system can only interpret the information it receives; it attempts to make adaptations to organize the level of the head with the rest of the body. Muscular facilitation and inhibition result, which may or may not create hypo- or hypertonicity, causing an additional symptomatic picture. In any event, additional receptors are stimulated and the effort of the adaptive process is continued. This, in essence, is what neurologic disorganization or "switching," discussed in Volume I, is all about. Applied kinesiology enables the examiner to find the primary disturbance instigating the chain of events. The key factor is that the physician understand the interactions of body function. For this reason it is extremely important that all physicians be aware of the manner in which the various areas of the body can influence other areas. This is true whether a physician limits his practice to a particular area of the body or not.

DISORGANIZATION WITHIN THE STOMATOGNATHIC SYSTEM



10—15. Proper afferent supply and integration equals balanced body organization. See text.



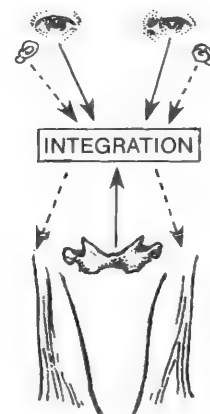
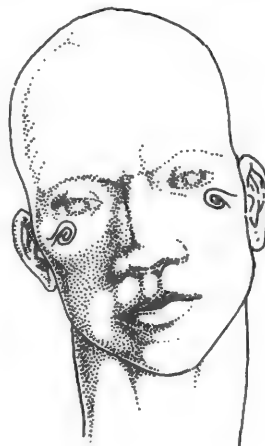
10—16. Improper afferent supply from visual righting reflexes creates an inability to organize with the other equilibrium proprioceptors.

The stomatognathic system is defined here as the shoulder girdle and all the structures above. This broad definition, related by Shore,⁴⁷ provides an excellent opportunity to observe the interactions of the body when one seemingly simple dysfunction develops. The model of receptor effects previously discussed is the basis for the interactions discussed now. We will use as an example the interactions of the equilibrium proprioceptors. Figure 10—15 illustrates afferent supply from the labyrinthine and visual righting reflexes and the body-on-head righting reflexes to the integrating centers for body organization. Disturbance in this organization can begin as a result of a change in the normal stimulation of the proprioceptors in the periodontal ligament. When centric occlusion is normal, the basic purpose of the proprioceptors in the periodontal ligament is to initiate the jaw-opening reflex upon tooth contact and to maintain the engram for jaw closing.

The proprioceptors are improperly stimulated from two types of disturbances: (1) malocclusion, where there is a prematurity or other factor that rocks or stresses a tooth, and (2) injury to the proprioceptor so that it causes impulses to be sent that are not in keeping with the actual status of the tooth. In either case, activity of the masticatory muscles changes. In the first case a new engram is built to change closing of the jaw to improve the occlusion. In the second instance a new engram may also be built, but not in keeping with the actual status of the occlusion. The muscular changes that develop may create cranial faults which may disturb function in any of the ways listed in Section I. For example, if a peripheral entrapment develops for cranial nerves III, IV, or VI, an ocular lock may develop, disturbing the

visual righting reflexes. If it is a vertical phoria, the visual righting reflexes send information indicating that the head is not level, when in reality it is. This causes the central nervous system to send efferent impulses to the primary head-leveling muscles — the sternocleidomastoid and upper trapezius — to level the head. If that happens, the head tilts to match the imbalanced level of the eyes. With the head tilt, the labyrinthine reflexes and the body-on-head reflexes send information that there is head tilt; yet the head tilt created from the vertical phoria causes the visual righting reflexes to send information that all is balanced.

Sound confusing? It is! The nervous system is in a turmoil trying to interpret conflicting afferent information. Efforts to balance the head are confused,

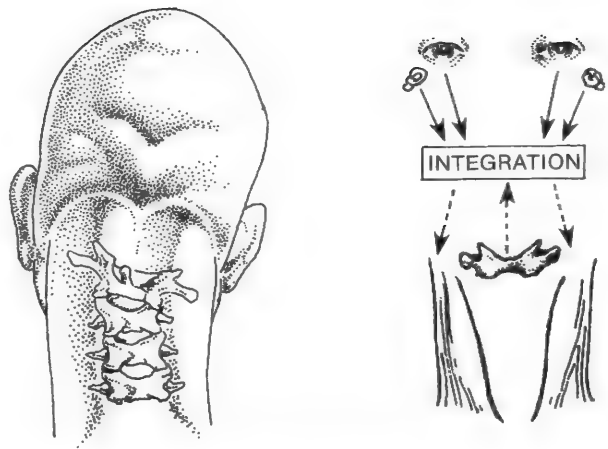


10—17. The afferent supply from the labyrinthine reflexes may be disturbed by nerve entrapment or by mechanical derangement of the temporal bones.

Cranial and Stomatognathic Area Interdependence

and the scenario goes on. Tension in the muscles will probably develop, and interaction of the muscle proprioceptors will be confused. It is possible that control of the hyoid muscles may be disturbed, influencing their apparent integrative role in body-on-head balance and in general equilibrium. There is a possibility that the anterior belly of the digastric may contract at inappropriate times during mastication and general mandibular movement. The interactions of the stomatognathic system are in a state of stress which may manifest symptoms anywhere within the system. This wide range of disturbance develops from what appears to be a very simple abnormal stimulation of the proprioceptors in the periodontal ligament.

Another example illustrates how stimulation of remote receptors can influence occlusion. A lateral subluxation of the atlas causes the vertebra to be superior on one side and inferior on the other as it follows the condyles on the occipital bone. This causes the receptors of the body-on-head reflex to interpret a head tilt. Again, disparate information comes from the labyrinthine reflexes and the visual righting reflexes, resulting in muscular stress. The afferent information is interpreted to facilitate and inhibit the sternocleidomastoid and upper trapezius muscles to balance the head. These imbalanced muscles pull into the cranial primary respiratory mechanism much the same as imbalanced muscles of mastication do. Cranial faults develop, and peripheral entrapment of cranial nerve V may result in poor integration of highly refined muscle coordination during mastication. The resulting malocclusion causes torsion and other types of strain on the teeth, stimulating the periodontal ligament proprioceptors. Interpretation of this afferent information results in an effort to develop a new engram of mastication. Again, neurologic disorganization takes place throughout the entire system, disrupting normal function.



10—18. Upper cervical subluxation or fixation causes improper afferent stimulation with resulting confusion in body organization.

Still another example is a cranial fault causing peripheral nerve entrapment of cranial VIII, changing the labyrinthine afferent impulses so they do not equate with the current head-leveling status. Again, with this information being integrated with varying impulses from the body-on-head and visual righting reflexes, confusion develops. It also appears that improper mechanical relation of one labyrinth to the other may create cranial faults where the temporal bones are counterrotated.³³

This discussion has been limited to the stomatognathic system in its broad definition. It should be obvious that influence on body function is not limited to this area; it will ultimately affect pelvic balance through the body-on-body righting reflexes and cloacal reflexes. Spinal distortion can develop, causing subluxations which in turn alter autonomic nervous system function from the sympathetic level. Potential influence is literally throughout the body.

Iatrogenic Conditions in the Stomatognathic System

The great amount of interaction that takes place in the stomatognathic system may cause a disturbance over a wide area. This is true with all aspects of the triad of health. The etiology most often develops on the structural side of the triad, with accidental and habitual trauma the most frequent causes. In addition to these, the possibility of iatrogenic problems must be considered not only as a cause, but also in the realm of prevention.

It is important to understand these interactions because, as mentioned so often, in diagnosis it is

impossible to recognize what you do not know. When a patient complains that particular symptoms developed shortly after the last treatment, the comment is often shrugged off by the physician with, "That problem is not related to what we've done; it's just a coincidence." As we develop greater understanding of the vast interrelationships of body function, we find that many of the associations patients make are indeed accurate. A few of the comments heard from patients are: "I've developed a pain behind my eye since the last neck adjustment."

"My neck hurts since my tooth was pulled." "I've had a ringing in my ears since you adjusted my pelvis." "I'm dizzy and my balance is poor since I got my new crown." Can these symptoms develop as a result of our therapeutic efforts?

The adjustment of an occipital or upper cervical subluxation or fixation can cause a cranial fault. In correcting an occipital subluxation, the skull is often contacted at the mastoid process, mastoid portion, or on the occipital bone, and the force is directed into the cranium in such a way that a cranial fault results. This can be avoided in nearly all cases by contacting the skull very broadly so the force is dissipated over a wide area of the temporal and occipital bones. Of course, it is best to have an accurate vector so that a minimal amount of force is necessary to obtain the correction. The vector is best obtained by the challenge procedure as described in applied kinesiology.⁵⁵

When the upper cervical area is adjusted it, too, should be analyzed and corrected with a very accurate vector of force. Adjustive thrusts that include a considerable amount of rotation can adversely influence the cranium because of the attachment of the dura to the axis and 3rd cervical vertebra. The force can be transmitted to the cranium through the dura and reciprocal tension membrane, with resulting cranial faults.

Cervical traction is responsible for the development of many cranial faults. The head halter, which is generally applied on the mandible and occiput, directs force into the skull which may create cranial faults. The problem may be more severe if malocclusion is present. Pressure on the mandible is transmitted in an imbalanced manner to the maxillae through the occlusion, and to the temporal bone through the temporomandibular joint. The potential for creating cranial faults is present whether the traction is motorized, intermittent, static, or manual.

Structural changes as a result of manipulation or other therapeutic approaches throughout the body can cause disturbance in the stomatognathic system. Maladjustment of the pelvis can cause cranial faults directly through the dural attachments. Most other maladjustments throughout the body influence the cranium on a secondary basis. Possibly the best evidence of remote change affecting the stomatognathic system is observed in the fact that changing a heel lift can affect occlusion.⁵²

There are many ways dental procedures can improve function of the stomatognathic system or, on the other hand, can create problems in the system. It should be recognized that procedures which are able to correct conditions in the stomatognathic system can also cause the same problems in

a normally functioning system. It may not even be necessary to change the occlusion. Holding the mouth stretched open during prolonged procedures can cause injury to the muscle proprioceptors and send improper afferent information which changes the organization of the muscles of mastication, perhaps adversely influencing the cranial primary respiratory mechanism.^{29, 46} Prolonged over-extension of the jaw during dental procedures can directly cause cranial faults because the mandibular elevators are attached to various leverage points in the cranial closed kinematic chain.

Apparently the most common iatrogenic problem is from some type of occlusal change. This may happen as a result of improperly equilibrating a prosthesis. If a new crown results in a prematurity, the initial reaction is stress in the cranium; this may result in cranial faults. A new engram of mandibular closing may be developed to eliminate the prematurity; however, the cranial fault may remain. If the cranial fault causes further disparity of the muscles of mastication by way of cranial nerve V, then new occlusal problems develop. The resulting vicious circle may never be able to correct itself until proper equilibration of the crown is accomplished and the cranial faults are corrected. Failure to equilibrate restorations from caries control may result in similar problems.⁵⁶

Pressure applied during various procedures can also be the etiology of various types of problems. Strong pressure applied in seating a crown may cause cranial faults. Pressure on the teeth during prophylactic cleaning or periodontal surgery may be responsible for a periodontal ligament receptor disturbance which does not return to normal and sends improper afferent information into the central nervous system for integration. Fortunately, most disturbances caused by pressure of this nature are temporary and are returned to normal by the body's own self-correcting and self-maintaining efforts.

Mechanical stress from extraction, especially a difficult one, may cause cranial faults.³² It is easy to observe how the mechanical strain from an upper extraction can disturb cranial function, but what about a lower extraction? The traction on a molar forces the condyle of the mandible into the mandibular fossa, forcing the temporal bone into an expiration assist position. Mesial leverage on the tooth increases the force into the temporal bone on that side and decreases it on the opposite side, causing a torsion in the skull.

Magoun^{32, 34} suggests additional stresses which may be possible in tooth extraction. A significant factor that may have been present in the past is the internal rotation of the temporal bone caused by the

Cranial and Stomatognathic Area Interdependence

V-type headrest in older style dental chairs. A patient will involuntarily force his head back into the headrest during an extraction. This is of even greater concern if the head is held firmly in the headrest during the extraction.

Possible influence of extractions on the cranial primary respiratory mechanism is emphasized by Henningsen, a dentist,^{22, 23} in his statement, "An awareness of the motion and function of the cranial structures enables the dentist to render a better service to the patient, and some perplexing dental problems will be better understood." Cranial faults are "... sometimes produced unknowingly during extractions through the use of elevators, forceps, and mouth props which can be powerful fulcra. Forces capable of delivering a tooth from its bony support are powerful and can be transferred via the articulations to areas distant from the mouth."

Chemical and organic changes can also influence the stomatognathic system in many ways. This follows the chemical influence on the structural portion of the triad of health. As is seen throughout applied kinesiology, nutritional deficiencies or adverse chemicals can cause either muscle hypo- or hypertonicity, disturbing balance of the structure supported by the involved muscles. This may be the first step in creating the vicious circle of interacting structures discussed in this chapter. A common example of chemical influence on the cranial mechanism is the antacid or hydrochloric acid influence on the temporal bulge cranial fault, discussed on pages 176 and 221. The mechanisms of this activity are unknown, but the clinical observation is a consistent one. It is probable that other medications, and possibly nutrition, have a similar influence on the cranial primary respiratory system.

How the mental side of the triad affects structure is well documented, especially in the stomatognathic system.^{29, 36, 41, 43, 44, 58} Most of the study in reference to the stomatognathic system relates with bruxism and bracing. Clenching and grinding of the teeth may be a reaction to stress during different activities of the day, or it may be nocturnal. Increased activity of the mandibular closers has been evaluated by portable electromyographic biofeedback equipment.^{43, 44} These studies have specifically shown that bruxing increases with emotional stress. When this muscular action is forced upon the masticatory system, the war — as usual — is lost by the bone and teeth, not the muscle. The problem is greatly intensified if there is malocclusion or muscular imbalance. Either of these problems can put imbalanced force into the cranium, which may create cranial faults. The major therapeutic effort in this case should be directed toward the cause of

emotional stress, which can be strictly on the mental side of the triad of health or — as discussed in Volume V — from the structural or chemical side.

When the mental side of the triad is involved, it does not appear that it is often induced by therapeutic efforts; however, the possibility of iatrogenic cause should be kept in mind. As we learn more about the mental processes, it appears that many mental health problems result from physiological disturbance. It is possible that physicians cause more mental health problems than is currently thought.

Sometimes the therapeutic effort itself is outmoded or potentially harmful. A physician must keep abreast of current research findings to apply the best possible approach in examination and therapy. With today's knowledge it is easy to determine the exact vector of correction necessary in manipulation of the spine or cranium. General massive manipulation is rarely indicated, and most often contraindicated. It is easy to challenge and determine exactly which vertebra is to be adjusted, and in which direction; the same is true in cranial manipulation. Each cranium has different morphology and interactions of the muscles, sutures, reciprocal tension membrane, and nervous system. Ramfjord⁴⁰ cautions the dental profession in reference to obsolete and sometimes harmful procedures. He refers to unnecessary temporomandibular joint surgery and the use of "sclerosing solutions" for dysfunctional disorders. He goes on to state that the problem includes "... a number of 'rehabilitation' and 'bite raising' procedures that patients may learn to live with, but which they would have been better off without."

No physician has the ability to treat every condition presented for examination. Because of this it is extremely beneficial to both the physician and the patient to have an understanding of body integration on a functional basis. This knowledge is often lacking because in modern health sciences specialization tends to be the trend; a physician's education is largely guided by his in-depth interest in a specialty. Too often a patient makes his own diagnosis when choosing a doctor to examine and treat his condition. If a patient chooses an oral surgeon to evaluate his condition, the treatment will likely be surgery. Choice of a specialist in equilibration will yield equilibration. A specialist in vertical dimension increase will yield a bite plane or splint, usually followed by more permanent prosthesis. Choice of a chiropractor may yield manipulation and attention to muscle balance, but failure of any occlusion or TMJ evaluation. Obviously the ideal approach is to find a physician knowledgeable in body interactions, how to find the basic underlying cause of the problem, and what therapeutic ap-

proaches are available for its correction. With this understanding proper therapeutic efforts can be made, whether or not they are in the examining doctor's field of expertise; if not, referral to other specializations can easily be made. This interaction between physicians yields improved patient treatment and is beneficial to all physicians involved. Possibly more important, it helps prevent iatrogenic

disturbances from lack of knowledge of how one treatment can adversely influence other areas of the body.

It is of value to re-state the importance of listening to the patient when he complains that a symptom complex developed after a specific treatment. The fact that we do not understand the correlation does not mean one is not there.

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Chapter 11

Temporomandibular Joint

General Description

The mandible and the temporomandibular joint in man are different from those in any other mammals or mammal-like reptiles.²⁰ In a horizontal animal, the jaw can swing down from a hinged articulation in an unimpeded manner. This is not possible in man because the head sits atop the spinal column rather than out in front of it. If there were no provision for the jaw to open except for the mandible to swing downward and posteriorly in a hinge-like action, it would impinge upon the trachea and esophagus, forcing them against the spinal column. Jaw function and its interrelationship with body activity appear to be unique to man. The articulation and its supporting structures, especially the muscles and nervous system, have a more complicated and critical activity than in any other mammal.

There is full differentiation of all articular elements of the temporomandibular joint by the fourth fetal month. A general embryogenetic law is that at this stage all vital organs have been formed.⁶ This seems to indicate the importance of the temporomandibular joint. The condylar cartilage of the articulation develops in a manner different from that of the long bones. It represents a specific growth center which develops independently from the skeletal cartilage primordium, which gives rise to epiphyseal plates and basic cranial synchondroses.⁶

There are many controversial factors about the temporomandibular joint in the literature. It is probable that much is still to be discovered about this joint; even currently known factors about the

anatomy and physiology need further investigation. One area of controversy has been whether temporomandibular joint dysfunction can influence auditory function and equilibrium. Phylogeny reveals that the middle ear and its structures important to hearing are elements of the primitive jaw joint. There is also a neurological connection between these middle ear structures and the muscles of mastication.²⁰ The mandibular-malleolar ligament was first pointed out by Pinto in 1962.^{24, 90} The tiny ligament connects the neck and anterior process of the malleus to the medioposterosuperior part of the temporomandibular joint capsule, the interarticular disc, and the sphenomandibular ligament. The ligament seems to be continuous with the tympanic membrane and has an embryologic origin common with that of the malleus and incus. Ermsar²⁴ states, "This tiny anatomical interassociation of the joint apparatus and middle ear may well explain many of the middle ear complaints described by Costen¹⁵ and others in the presence of temporomandibular dysfunction." Pinto points out that more research on this association is needed. It seems odd that the ligament, described twenty years ago, is still not listed in the recent general anatomy texts.^{45, 139}

The temporomandibular joint does not take on the anatomical description of an adult until considerable development has occurred. Formation of the articulation is in response to the growth of adjacent structures, including the outward growth of the middle cerebral fossa and the fusion of the tympanic

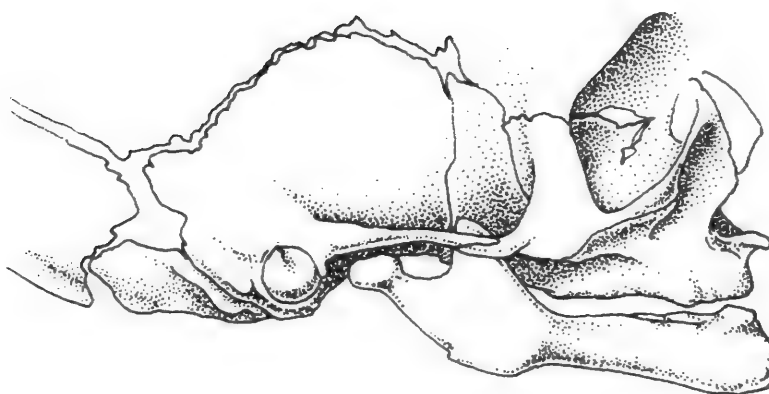
Temporomandibular Joint

ring, along with development of the external auditory meatus.⁷⁸ The newborn has a functional temporomandibular joint, even prior to the development of the surrounding structures comprising the fossa. These are the external auditory meatus, fusion of the tympanic ring, and the articular eminence. Without an articular fossa, the mandibular activity for suckling depends totally on the balance of the masticatory muscles. This makes normal function of cranial nerve V very important at this stage of life. Its entrapment by cranial faults can cause imbalance of the masticatory muscles, producing improper joint function and possibly maldevelopment. Cranial faults can also create temporomandibular joint disturbances by poor mechanical relation of the right and left TMJ, such as counter-rotation of the temporal bones, creating asymmetry of the mandibular fossae.⁶⁷ The potential for future problems is even greater, since the shape of bone depends on muscle balance and its attachment.^{11, 76, 79, 108, 130}

Regardless of a physician's discipline, it is essential that he have knowledge of the intricate activities of the temporomandibular joint, whether he be a dentist dealing with occlusion or an applied kinesiologist dealing with cranial faults and muscular balance. The joint, its soft tissues and muscular

kinesiology, and the attending neurologic control make up the TMJ complex. It is not possible to obtain a knowledge of this articulation by study of the bones alone. This can easily be observed by fitting the mandible to a dry skull. When the teeth are in intercuspation there is no bony contact at the temporomandibular joint. To make a hinge action at the joint in a dry skull, it would be necessary to disocclude the teeth. This is a highly complex structure requiring the presence of the articular disc for an anatomical study.

A working knowledge of the joint, neurology, muscles of the hyoid, skull, and mastication is necessary to appreciate TMJ function and occlusion. This working knowledge is integrated with adjacent complexes, such as the cranial primary respiratory and hyoid mechanisms. The larger complex is then integrated with the closed kinematic chain of the head-to-shoulder muscles, creating the stomatognathic system in its wider definition. The workings of this system are finally integrated with the rest of the body. An intricate knowledge of the muscles and their importance is pointed out by Ricketts⁹⁷ in his statement, "Disturbances of the temporomandibular joint are almost always associated with muscular disturbances."



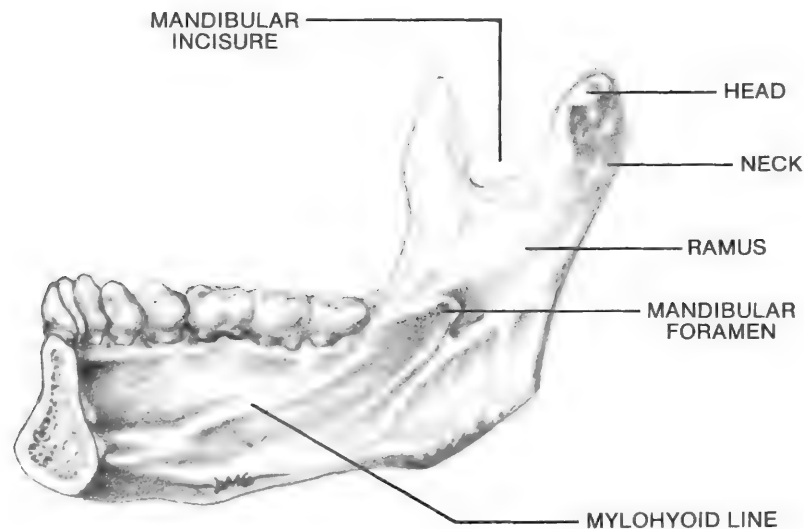
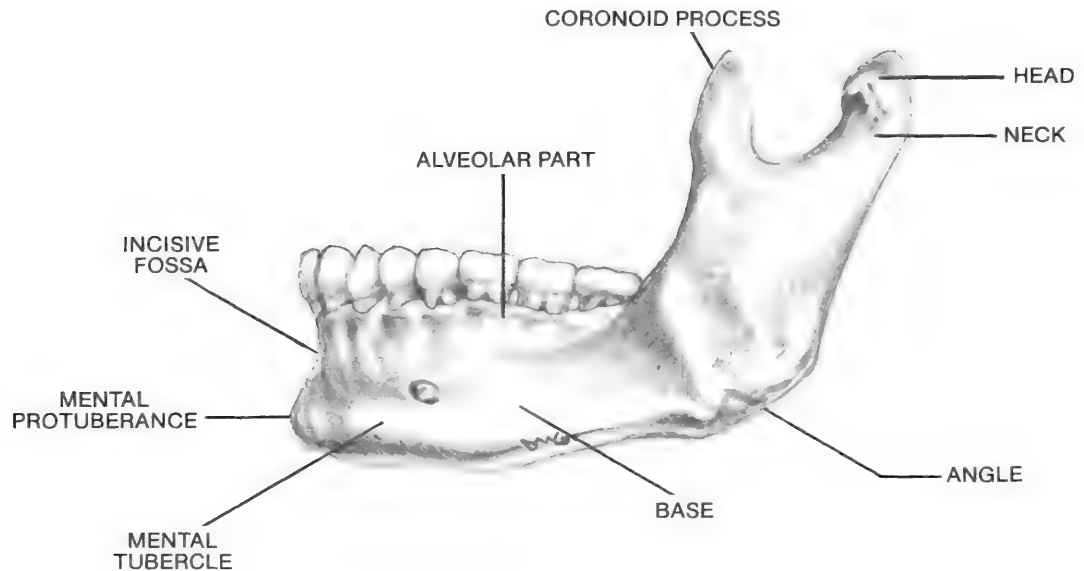
11—1. Absence of an articular fossa in an infant creates even greater demand for balance of the masticatory muscles.

Anatomy

MANDIBLE

The mandible is composed of a U-shaped body and two broad rami projecting superiorly from the posterior ends of the body. The body of the mandible is marked on the exterior surface with a midline — the symphysis menti — representing the fusion of the two halves of the fetal bone. At the inferior aspect of this ridge is the mental protuberance. The mentalis muscle originates above the protuberance at the incisive fossa. The mental foramen, through which

pass the mental nerve and vessels, is below the second pre-molar. The lower border of the body is referred to as the base of the mandible. The anterior portion of the digastric muscle attaches close to the internal midline of the base. The upper border of the body is composed of the alveolar sockets. The internal surface of the body is divided into two sections by the mylohyoid line, a very heavy line in the molar region which becomes smaller at the



11—2. Mandible.

Temporomandibular Joint

anterior portion of the body.

The ramus of the mandible is quadrilateral with two surfaces, four borders, and two prominent processes. The inferior border is continuous in front with the base of the mandible. Posteriorly, it rises from the inferior border of the base to form the posterior border. The joining of these two borders is called the angle of the mandible, where the bone generally flares laterally. The posterior border extends to the condyle of the mandible. The anterior border arises from the alveolar part of the base and ends above as the coronoid process, which is the point for insertion of the temporalis muscle. The superior border forms a wide notch — the mandibular incisure — between the coronoid process and the condyle. The lateral border is flat and flares laterally at the angle of the mandible. The medial surface presents the mandibular foramen, through which pass the inferior alveolar vessels and nerve.

As the mandible enlarges with age, the relative position of the body and rami changes. The change in shape of the mandible is on a predictable arc of a circle. The predictability of mandibular growth was demonstrated by Ricketts,⁹⁸ who developed and

plotted an arc of mandibular growth from lamina-graphic and computerized cephalometric study. Orofacial growth must meet the mandibular change. These factors are regulated to a certain extent by balance, or lack of it, from muscular pull.

As discussion of temporomandibular joint function proceeds, it is important to recognize the great variations present in individual mandibles. Although basic descriptions of actions are presented here, each evaluation is individual. Ricketts⁹⁴ studied 200 temporomandibular joints with cephalometric laminagraphy. His first comment in the discussion of the study was, "Probably the most arresting concept gained from this study is the great range of variation found in practically every aspect investigated." His final comment was, "The evidence seems to indicate that too much emphasis has been placed in the past on the mechanics of the joint, i.e., on condyle-fossa relationship. The opinion expressed by Robinson¹⁰³ viz., that the **muscles** are almost solely responsible for the intricate movements of the mandible, seems to be more in accord with observations of the functioning joint." They are also very important in the growth and function of it.

TEMPOROMANDIBULAR ARTICULATION

The articular joining of the cranium and the mandible is usually referred to as the two temporomandibular joints. Different terminology is sometimes used. *Sicher's Oral Anatomy*²² refers to the articulation as the craniomandibular joint, with reference to the action between two rigid components, the skull and the mandible. Even though in applied kinesiology we recognize that the skull is not a totally rigid component in the activity, the argument has validity; these thoughts should be kept in mind during study of the temporomandibular joint.

The mandible is a solid component and has only the micro-flexibility of living bone. When the temporomandibular joint moves on one side, there is some type of activity on the opposite. It may be minimal, or the majority of activity may be on the opposite side. *Sicher's Oral Anatomy*²² points out that the incorrect concept of considering the TMJ on one side only is often initiated in anatomy laboratories where a cadaver is sagittally sectioned; a student observes the individual action of a temporomandibular joint since the mandible has been severed. Keeping in mind that when one temporomandibular joint moves there is some activity on the opposite side, we will

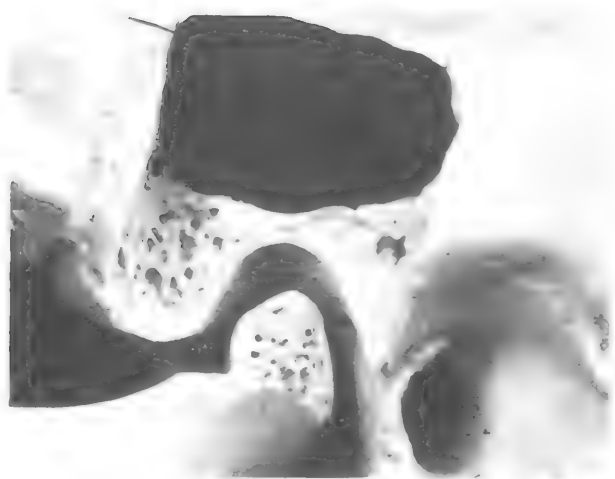
continue to use the more popular term — temporomandibular joint.

To understand the action and purpose of the temporomandibular joint, it is necessary to understand the histology of the mandibular fossa and the condyle. The primary consideration in reviewing the histology is to determine whether the articulation is designed for weight bearing and the attendant stress that accompanies it. There is disagreement about the purpose of the joint in mandibular activity. Some authorities assert that the action is that of a third-class lever, while others vigorously argue that it is not. The disagreement is probably due to the fact that this extremely complicated joint has numerous actions with different purposes. It is a joint complex that is unique in the body, as are its histology and interdependence with other structures and activities. Motion and lever action will be discussed later.

The temporal bone has previously been discussed (page 53). A closer look at the mandibular fossa (glenoid fossa, articulating fossa) with its histology is of value here. In referring to the parts of the fossa, there is a discrepancy in terminology by various authorities. The terms used here have been



11—3. Teeth in centric occlusion.



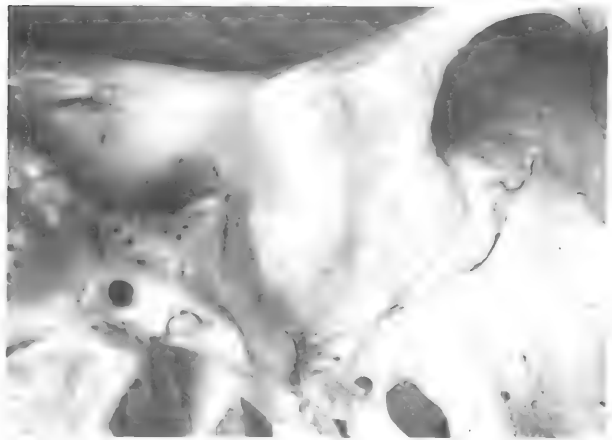
11—4. Section into the condyle and glenoid fossa.

chosen to aptly describe the structures and their respective purposes.

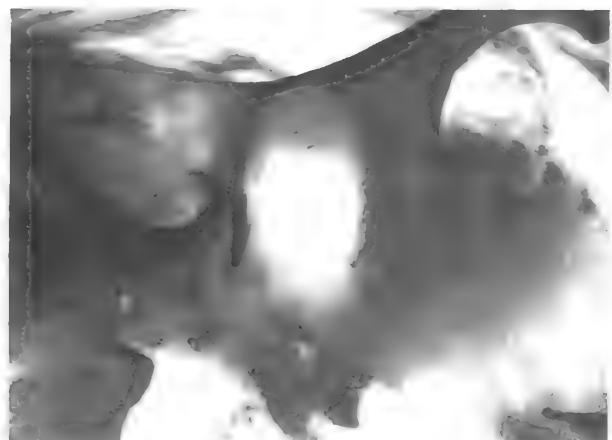
The fossa is located at the base of the zygomatic process of the temporal bone. It is smooth, oval, and deeply concave, formed by the squamous part of the temporal bone. It is bounded posteriorly by a small conical eminence called the postglenoid tubercle, and anteriorly by the articular eminence. Anterior to the eminence is the articular tubercle, which is non-articulating²² and provides attachment for the capsular ligament. Posterior to the mandibular fossa is the separation between the squama and tympanic sections of the temporal bone, known as the tympano-squamosal fissure.

There is considerable variance in fossa shape; some are very shallow, others deep, some small, and others large.⁹⁶ The bone separating the mandibular fossa from the area of the temporal lobe of the brain varies somewhat but is mainly very thin, with an absence of cancellous bone. This indicates that this area is not meant to receive great stress, because cancellous bone covering a dense cortical plate is considered as a weight-bearing and stress-receiving structure.¹⁰³

The mandibular fossa and articular eminence are entirely covered by a layer of fibrous tissue. It is thickest on the articular eminence, and very thin on the mandibular fossa. This seems to give added support to the theory that the fossa is not designed to be a weight-bearing articulation. In addition, the soft tissue is not the type designed for weight bearing.¹¹⁵



11—5. Inferior view of right glenoid fossa.



11—6. Transillumination through the right glenoid fossa as pictured in 11—5. Note the thinness of the bone.

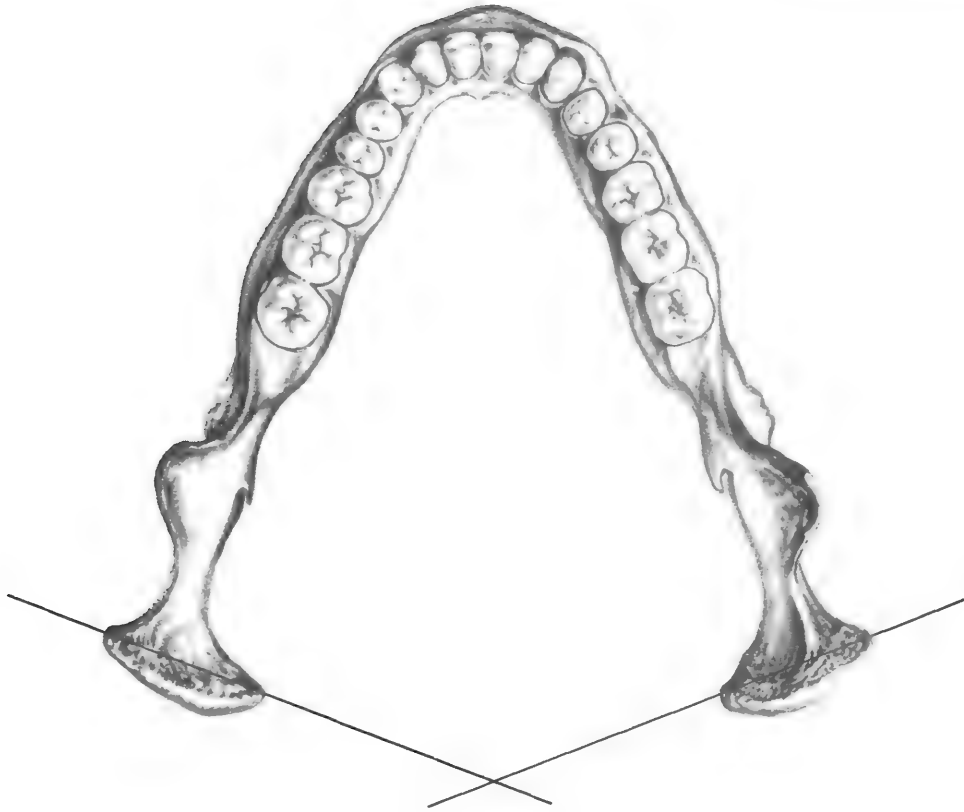
Temporomandibular Joint

The mandibular condyle is a convex, ovoid enlargement at the superior aspect of the rami of the mandible. The entire enlargement is called the head of the mandible. The constricted portion immediately below the head is termed the neck. The ovoid condyle has its long axis generally in the lateral transverse plane. Lines drawn through the lateral axis of each condyle meet at a point posterior to the condyle, because the condyles are usually at right angles to the axis of the main portion of the body of the mandible.¹³⁵ Ricketts¹⁰¹ has standardized on a 20°

central ray for the oblique section of the TMJ to accommodate this angulation. When lines are drawn through the long axes of the condyles, they intersect at the basion which is the anterior border of the foramen magnum. The basion is the pivot center of the head on the cervical spine. It is, therefore, the area of least motion with head flexion, extension, and rotation, creating the least stress on the meninges. This relationship seems to indicate a functional coordination between the joints of the mandible and the head on the cervical spine.¹⁰¹



11—7. Lines drawn through the long axes of the condyles intersect at the basion. This is an area of minimal movement during flexion, extension, and rotation of the head on the neck.



11—8. Note that the condyles are not in alignment with each other. The position of the long axis of the condyle is close to 90° with the long axis of the ramus and body of the mandible.

Condylar shapes vary considerably. They are rounding, flattening, flattening with overhanging lateral and medial slopes, marked lateral slope flaring and flattening, marked medial slope, downward flattening of both slopes, thin condyle with backward flattening, and a more or less marked depression of the posterior part. This variance is due to remodeling as a result of pressures applied to the joint by different types of occlusion which cause pathological stress.⁷⁵

The nature of the bone and its fibrous covering mentioned above supply evidence that the joint is not designed for weight bearing, but this idea is very controversial. Before this subject can be discussed, the anatomy and histology of the articulating disc must be understood. It is impossible to understand the temporomandibular joint by studying a dry skull because of the role of occlusion and the disparity between the articular surfaces. This difference in shape is accommodated by the disc to make a working articulation. The morphology of the disc varies greatly, as the considerable discrepancy in the literature regarding its description emphasizes. The usual description¹³⁹ is that of a roughly oval plate

consisting of fibrous tissue. Its upper surface is concavoconvex, accommodating to the form of the mandibular fossa and articular eminence. The inferior surface is concave, accommodating the surface of the mandibular condyle. The circumference is connected to the fibrous capsule, and in front to the tendon of the external pterygoid muscle. Each disc is attached to the mandibular condyle by strong fibrous bands which are responsible for their simultaneous movement in normal function. Posteriorly the disc attaches to the margin of the mandibular fossa.

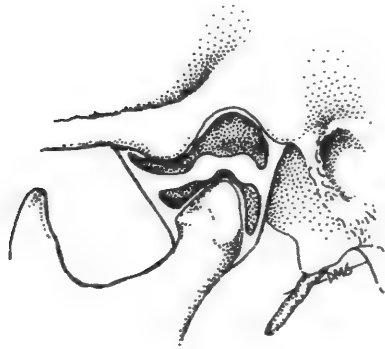
After the fifth decade of life, microscopic evidence of degeneration is observed with fraying, thinning, and perforation to a varying degree. This is observed so often that it is considered a “normal” aging process by some. Wakeley¹²⁸ takes exception to this description, stating that the disc is irregular, being thicker in the center and anterior portions and having a distinct depression between these areas. The posterior part of the cartilage is very thin, fusing behind with the capsule. No perforated discs were found in his examination of over fifty specimens.

Ricketts¹⁰¹ laminagraphic experience indicates the joint should be studied at three levels — the

Temporomandibular Joint

lateral, central, and medial aspects — because the anatomy and wear differ considerably. The major wear area is at the lateral aspect, which becomes thinner than the medial area. The serious thinning and perforation is at the central area. The different wear levels are due to incongruity in joint movement during different mandibular actions.

There is a potential cavity above and below the disc, separating the temporomandibular joint into two movable areas. It is generally considered that these compartments are lined with a synovial membrane. The “spaces” are potential ones only, with the gliding actions supplied by the lubricating factor of the synovial membrane. The inferior cavity is the smaller, and it is here that the hinge action takes place. The gliding action of the condyle over the articular eminence takes place at the larger upper cavity; in other words, the disc moves with the condyle.



11—9. *Sagittal section of the TMJ, with the potential cavities opened for demonstration.*

The articulating surfaces are covered with fibrous connective tissue. Occasionally some cartilaginous cells are reported in these tissues. It is generally considered that if they are present at all, they are few in number. The fact that these articulating surfaces are covered with fibrous connective tissue rather than cartilage is considered by many to be significant regarding the purpose of this joint. Cartilage is an excellent tissue for a weight-bearing joint because it resists the forces of compression, as well as presents a smooth surface for the articulation; on the other hand, it is not very resistant to the forces of shear. Moss⁷⁷ reflects on the fact that the temporomandibular joint does not work as a single entity but is, in reality, two joints; in the lateral movements of the mandible there are inevitably considerable compo-

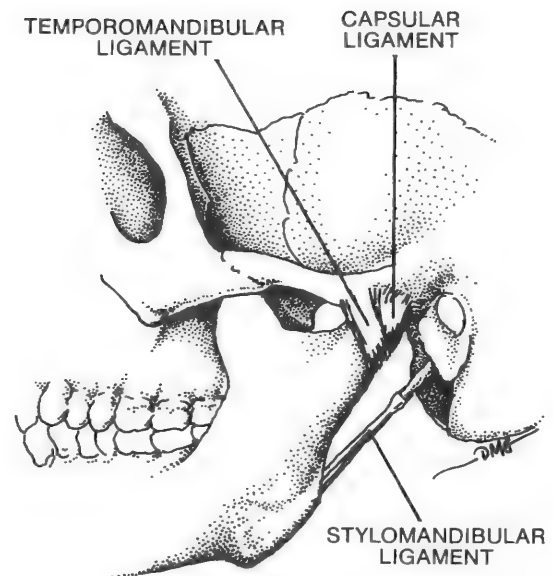
nents of shear imposed on the joint surfaces. Fibrous connective tissue is particularly well-suited to withstanding these forces. In the depths of the mandibular fossa, where there is no sliding activity as such of the joint, the fibrous tissue is very thin.

Many stress that the thinness of both the fibrous tissue and the bone, plus lack of cancellous bone in the superior portion of the mandibular fossa, is strong evidence that the temporomandibular joint is not designed for weight-bearing forces;^{22, 103} yet others suggest that the articulation is strong and capable of weight bearing. Shore¹¹³ relates to the compact bone which, in the area of the articular eminence and condyle, is underlined by cancellous bone, giving the bone weight-bearing characteristics. He adds that “. . . the fibrocartilage increases in thickness as it continues from the glenoid fossa to the posterior wall of the articular eminence. Evidence of inherent resistance to stress is found in the characteristic arrangement of the fiber bundles in the fibrous coverings. The surface fibers are parallel with the surface; the deep fibers are perpendicular to the surface. This arrangement is similar to that found in other articular cartilages. The surface layers are adapted to gliding while the deep ones are adapted to the resistant force.” Dawson¹⁸ refers to the condyle as being “. . . truly a stress-bearing joint and the disc that is interposed between the condyle and the eminence is also made to bear stress. It is tough fibrocartilage, and the fact that it is avascular in its stress-bearing area is proof that it was intended to be a stress-bearing joint.” Analysis of the biomechanics of the temporomandibular joint indicates that it is load-bearing during function.³ Probably both weight-bearing and non-weight-bearing philosophical views are correct. Proponents of the view that the joint is a non-weight-bearing one are primarily those who deal more with increasing vertical dimension to remove stress from the temporomandibular joint. Those who contend that the joint is weight-bearing deal more with occlusal equilibration as a mode of therapy. Both of these views will become clearer as we proceed with additional chapters in this text. For now it will suffice to state that there is no single optimum approach for all cases of temporomandibular joint disturbance. Each individual has to be studied and all aspects of the stomatognathic system evaluated. When the various problems that develop within this system are studied, the question of the joint being a weight-bearing one or not resolves itself. With normal occlusion, the articulation appears to have adequate strength for any activity required of it. There appears to be enough reserve strength in the joint to manage some loss of vertical dimension, but it is limited in severe cases.

Ligaments

The temporomandibular articulation is maintained in its position primarily by the mandibular muscular sling and other controlling muscles. The ligaments' primary purpose appears to be limiting the maximum range of joint motion. Moss⁷⁷ goes so far as to say that "... it may be stated that the ligaments of the temporomandibular joint do not restrict the normal range of motion and that the ligaments are not necessary for the functioning of the joint. Muscle forces are sufficient to approximate the joint surfaces and the ligaments are not brought into play during the normal excursive motion of the joint." DuBrul²² partially agrees in stating that "... the sphenomandibular and stylomandibular ligaments (have no) influence on the movements of the mandible." This discussion is in reference to normal movement. It should be recognized that ligaments limit excessive movement, especially in the presence of muscular imbalance where the ligaments are important in preventing dislocation.⁶¹

The temporomandibular joint has four ligaments generally described in the literature: the capsular, temporomandibular, sphenomandibular, and stylomandibular. The *capsular ligament* has four portions completely surrounding the condyle. The anterior portion attaches to the articular tubercle immediately in front of the articular eminence. The posterior portion attaches to the squamotympanic fissure; the medial and lateral portions attach to the circumfer-



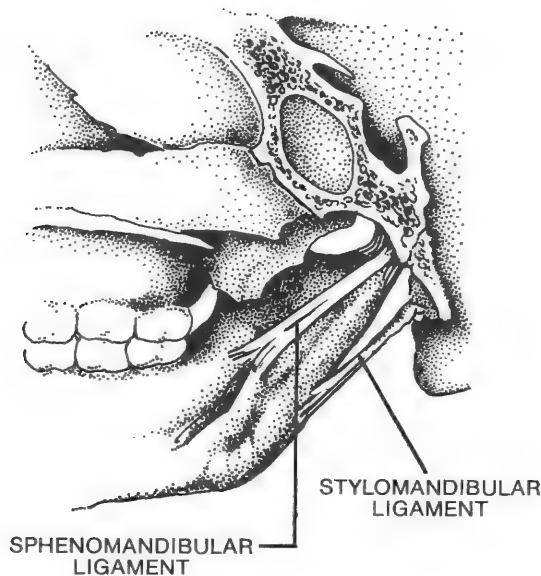
11—11. Lateral ligaments of the TMJ.

ence of the mandibular fossa. The inferior attachments of all portions of the ligament attach to the neck of the mandible. The fibrous capsule is lined with a synovial membrane, and the internal portion of well-defined fibers attaches to the inner edge of the mandibular fossa above and the inner side of the neck of the condyle below.

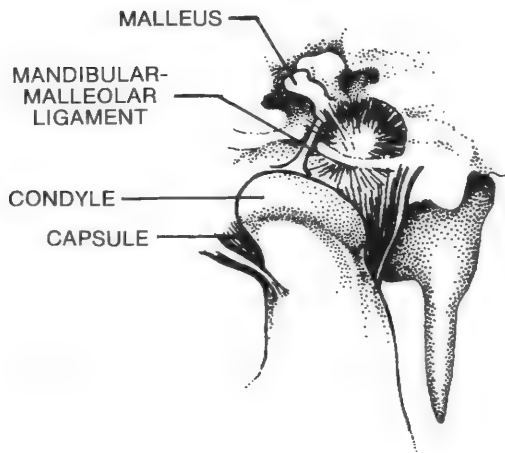
The *temporomandibular ligament* is classified as part of the capsular ligament by some⁷⁷ because it is a heavy thickening of the capsular ligament. It broadly attaches to the zygomatic process of the temporal bone above, with the anterior fibers attaching beyond the articular eminence. During hinge movements of the articulation, the ligament can act as a stabilizing factor; during translation, it becomes lax.

The *sphenomandibular ligament* attaches above to the spine of the sphenoid. It broadens as it descends to attach to the lingula of the mandibular foramen. Moss⁷⁷ considers the sphenomandibular ligament a collateral ligament (one which neither tightens nor loosens during joint movement) because the axis of rotation of the mandible during opening is at the lingula of the mandible.

The *stylomandibular ligament* originates from the styloid process above and attaches to the angle and posterior border of the mandibular ramus. It is considered an accessory ligament, and its functional status is uncertain. It is a specialized band of the deep cervical fascia.



11—10. Medial ligaments of the TMJ.



11—12. Mandibular-malleolar ligament (modified from Pinto,⁹⁰ *Journal of Prosthetic Dentistry*, Vol. 12, No. 1).

The mandibular-malleolar ligament is not described in the usual anatomy texts, but it has been demonstrated by Pinto⁹⁰ and others. It is a very small ligament which attaches anteriorly to the capsule, disc, and sphenomandibular ligament. It attaches posteriorly to the neck and anterior process of the malleus. This tiny ligament has a common origin in the embryological stage with the malleus and incus. This ligamentous attachment may be responsible for some of the symptoms that Costen¹⁵ observed in conjunction with temporomandibular joint dysfunction. Although Costen's basis for auditory malfunction with TMJ disturbance has been proven anatomically incorrect,^{105, 109} there are often clinically observed changes in auditory function after correction of temporomandibular joint malfunction. Other hypotheses have been presented for these symptomatic improvements.^{1, 2, 10, 36, 39, 66} No intensive study of this tiny ligament's role in auditory problems is found in the literature.

Classification of Condylar Position

Ricketts^{96, 97} has classified the position of the condyle in the fossa with the teeth in centric occlusion by laminagraphy. The average condylar position from the condyle to the eminence is 1.5 mm plus or minus 0.5 mm; from the top of the condyle to the mandibular fossa, 2.5 mm plus or minus 1.0 mm; and from the center of the external auditory canal, 7.5 mm plus or minus 1.5 mm. These measurements are corrected from the laminagraphic projected enlargement to represent the actual position of the condyle.¹⁰¹

Current methods in laminagraphy use a 20° oblique projection for accommodation to the angle of the condyle. There are standardized measurements which take into consideration the 6% enlargement of the projection.¹⁰¹

In studying the temporomandibular joint with laminagraphy and applying these measurements, care must be taken to allow for the amount of variable TMJ morphology. With this consideration, Ricketts defined four classifications of TMJ disturbance that can develop as a result of malocclusion. More recently he has named the four classifications, which are represented by initials.¹⁰¹

Type I (ANT — anterior displacement): An abnormal range of forward movement characterizes Type I; it is a result of excessive range of function and abnormal functional position of the condyle. Close examination on the laminagraph may show subchondral sclerosis on the anterior border of the condyle, and the joint spaces may be starting to thin out on the anterior border; this is characteristic of Angle Class II, Division I malocclusion. The problem develops because an individual must thrust his jaw forward to use the incisors. In some cases the involvement is caused by orthodontic appliances which position the mandible forward. From an applied kinesiology viewpoint, the external pterygoid is usually hypertonic, with the neuromuscular spindle cell probably disturbed from excessive use of the muscle to pull the mandible forward for incising. In some cases a patient must thrust his jaw forward in speaking and may habitually hold it forward for cosmetic and aesthetic reasons to gain closure of the lips. Although muscular treatment is important, the condition will return if other measures are not taken. Orthodontic treatment is usually required for a permanent solution.

Type II (DFD — distal functional displacement): Distal displacement, which is a posterior disposition, is controversial, but roentgenograms reveal that the condyle may be in direct contact with either the postglenoid process or the tympanic plate. This is often, but not always, associated with Angle Class II, Division II malocclusion. As the mandible moves from the rest position to occlusion it is driven backward by the contact of the incisors. Loss of the lower first molar and a shifting of the remaining teeth forward will cause the condyle to shift posteriorly and deeper into the fossa.

From an applied kinesiology standpoint, the disturbance caused by central incisor contact moving the mandible posteriorly requires orthodontic treatment for a permanent correction. Loss of the first molar and consequent slight loss of vertical dimension require critical evaluation (discussed in Chapter 12).

Type III (CLI — contralateral interference):

This type is observed on the laminagraph in its advanced stage by breakdown of the condyle and eminence resulting from trauma. It is due to malocclusion, which can develop from any cause. Already mentioned has been a particularly problematic situation wherein the occlusion is equilibrated to imbalanced skull activity; subsequently another physician corrects the skull or it corrects itself. Now there is malocclusion, which will probably re-create the cranial faults, and a vicious circle begins. Any type of change, such as the loss of teeth, can cause others to shift or supererupt, creating abnormal contacts. Whatever the reason for the malocclusion, the neuromuscular system is disturbed; this simply propels the problem into other structures and systems (see Chapter 10). The corrective approach is dental restorations and/or equilibration, as well as attention to the cranial primary respiratory system.

Type IV (LPS — Loss of posterior support):

Loss or retrusion of posterior teeth causes the condyle to be positioned superiorly, because forces

generally taken by these teeth are transferred into the temporomandibular joint. The condyle will be superior and anterior on a laminagraph because when there is a loss of posterior teeth, the anterior teeth are more active in chewing and the internal pterygoid and masseter become hypertonic, throwing the condyle superiorly and anteriorly. In this condition, the applied kinesiologist should evaluate the jaw-closing muscles very carefully and make appropriate corrections. If the condition is actually caused by a loss of vertical dimension, it must be increased by restorations or splinting. Before concluding there is a loss of vertical dimension when the natural teeth are present, a very critical analysis must be made (see Chapter 12).

The frequency of these conditions seen in an orthodontic practice is as follows:¹⁰¹

- DFD — Distal functional displacement
- LPS — Loss of posterior support
- CLI — Contralateral interference
- ANT — Anterior displacement

Mandibular Positions

Mandibular position refers to the relationship of the lower dental arch with the upper, and of the condyle in the mandibular fossa. There are three basic positions in the mandible's relationship with the skull: (1) the resting position of the mandible with the skull when the muscles are inactive, (2) when the teeth of the upper and lower arches are in complete articulation (intercuspatation), and (3) the position of the condyles in the mandibular fossae, which is called centric relation and has many definitions. In addition to considering the mandible's position with the skull, the bony structure should be related to the muscular structure. These relationships are very important to occlusal equilibration, TMJ function, fitting prostheses (dentures and crowns), and treatment devices (splints and bite planes). There is considerable controversy in the literature regarding the importance of the various relationships, and even about how to measure a relationship and record it. The controversy may be present because of the dynamic interrelationship of the TMJ with the rest of the stomatognathic system and the total body.

In applied kinesiology it has been observed that masticatory muscles function differently in various postures and at different phases of respiration; they are also influenced by various physical activities. The

sections of the closed kinematic chain vary in their activity with certain head positions. The change of masticatory muscle function with different head positions has been demonstrated electromyographically by Funakoshi et al.³⁷ An occlusion also varies with force applied to the skull, such as resting against a dental chair headrest.¹¹⁹ When researchers and authors establish "normals" for the various mandibular positions and discuss them, they should define terms to clarify whether variables have been taken into consideration.

Any physician reading this text should be aware of the importance of rest position, freeway space, centric relation, and centric occlusion to a dentist, and how the various muscular balances worked with in applied kinesiology relate with these positions. Any one or all of the mandibular positions can change when there is either localized or remote muscular imbalance of the type with which applied kinesiology deals. Cranial faults can disturb the nerve supply to the masticatory, hyoid, orofacial, or head-leveling muscles. Remote problems, such as abnormal stimulation of the foot proprioceptors, pelvic imbalance, spinal distortion and subluxations, and imbalance of the general postural muscles, can directly or indirectly affect mandibular position.

Temporomandibular Joint

Many dentists have observed that when there is an apparent leg shortness, a heel lift placed to compensate for it changes the occlusion. This was first observed in an electromyographic study of an individual with a short leg. When the compensating heel lift was worn, the muscles of mastication had a firing pattern of normal occlusion. When the heel lift was removed, the firing sequence was markedly altered during chewing.¹²³ Change of the EMG pattern of chewing is not always observed upon

standing when there is a short leg; however, it is not uncommon.

Although the heel lift is a device used by many, it is observed in applied kinesiology that placement of a heel lift often only compensates for a pelvic distortion¹²⁹ which should be corrected. Most often the factor which causes a change in the EMG upon standing is foot, pelvic, spinal, or some other dysfunction affecting the stomatognathic system's balance.

REST POSITION

The mandible is in the rest position when the musculature influencing it shows minimal activity. This includes not only the masticatory muscles, but all the muscles in the closed kinematic chain of the stomatognathic system previously discussed. This means that as an individual's posture changes, the rest position also changes. For example, an individual with increased anterior to posterior spinal curves is in a position of cervical spine extension. This stretches the hyoid muscles and platysma, which will increase the dimension of the rest position to bring equilibrium between the stretched muscles and the mandibular elevators. On the other hand, if the neck is flexed and head nodded forward, the tissues under the mandible are bunched up; this pushes the mandible upward to decrease the rest position. The rest position is also influenced by the body's orientation in space. When supine, the mandible is in a more retruded position; as the body moves into a more upright position, with no change in the relationship of the head to the shoulder girdle, the mandible shifts forward.⁷¹

To be in a true rest position, muscles must be inactive. The position in which an individual habitually holds the mandible may not be a true rest position. Abnormal function or improper habits create habitual mandibular postures. An individual with tongue thrust and weak lips may be a mouth breather who habitually holds the mandible in an open position. In these cases there is often excessive activity of the muscles in what appears to be the rest position; in reality, it is an habitually contracted position of the jaw-opening muscles. On the other hand, clenching of the teeth caused by emotional factors elevates the mandible, giving an habitual closure position. Habits such as pipe smoking, toothpick chewing, etc., take an individual away from the rest position, again creating habitual closure. In all these activities there is interference with the rest position; the excessive

muscular contraction causes an imbalance in the closed kinematic chain.

When there is malocclusion, the rest position is unstable. This is corrected with equilibration.⁸⁷ Instability of the rest position as observed on the Mandibular Kinesiograph, explained later, is also corrected by applied kinesiology methods.

Prior to determining the rest position, it is necessary to have normal muscle function throughout the stomatognathic system, not just in the muscles of mastication, because of the interaction of the closed kinematic chain. Applied kinesiology techniques are used to evaluate the muscles of the stomatognathic system and those remote in the body. After making corrections, it is often observed that the apparent rest position changes, usually increasing.

Carlsöö¹² found that the most important muscle in the habitual closing movement of the mandible appeared to be the temporalis. He also stressed the temporalis' importance in the habitual rest position. This is an important muscle to evaluate with applied kinesiology techniques when there is instability of the rest position.

A system of transcutaneous electrical neural stimulation (TENS) has been developed by Jankelson to induce masticatory and facial muscle relaxation.^{53, 54, 55, 56} The neural stimulation is generated from a specially designed unit called the Myo-monitor.⁸⁴ The transcutaneous stimulation is to cranial nerves V and VII by electrodes placed anteriorly and inferiorly to the ear. The muscles are pulsed just to the point of contraction, making certain that the muscular contraction does not cause tooth contact. The stimulation is done for thirty to forty-five minutes, after which measurement of the rest position generally indicates that the muscles have lengthened. The rest position established by this method does not necessarily parallel the rest

position established following applied kinesiology muscle balancing techniques.

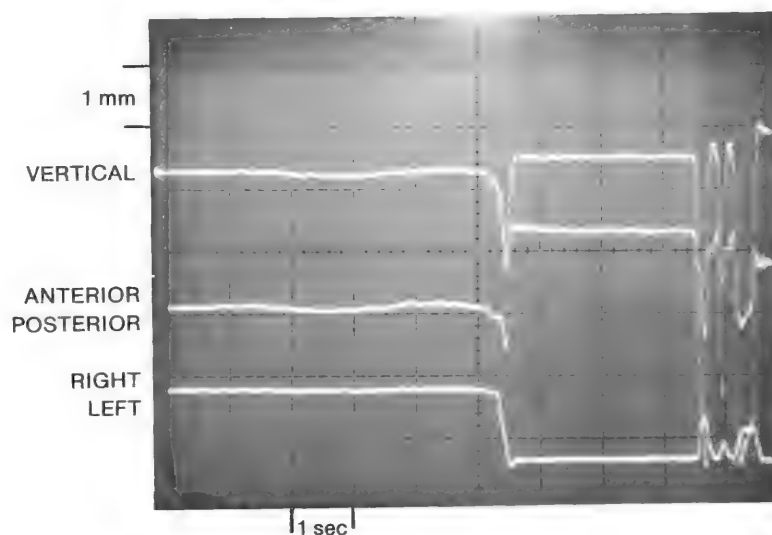
Standard methods which are reproducible should be used for the measurement. The mandible's normal rest position must be thought of as an active process since it changes to meet the current status of the entire complex.¹⁰³ Not only does the rest position vary with changes in the dentition,⁸⁵ it will also vary with an individual's postural changes, which influence the stomatognathic system.

Measurement of the rest position is usually done with a patient sitting upright, with the Frankfurt plane horizontal. The best method is to observe muscle

activity by electromyography. This is usually accomplished by placing surface EMG electrodes over the mandibular elevators to observe when there is electrical silence, indicating muscle inactivity,^{88, 114} this is considered the ideal rest position. In a normal individual, repeated measurements using this method will have reproducibility if the parameters of the test remain the same. There is poor reproducibility when malocclusion is present, but reproducibility of the rest position develops after proper equilibration.⁸⁸

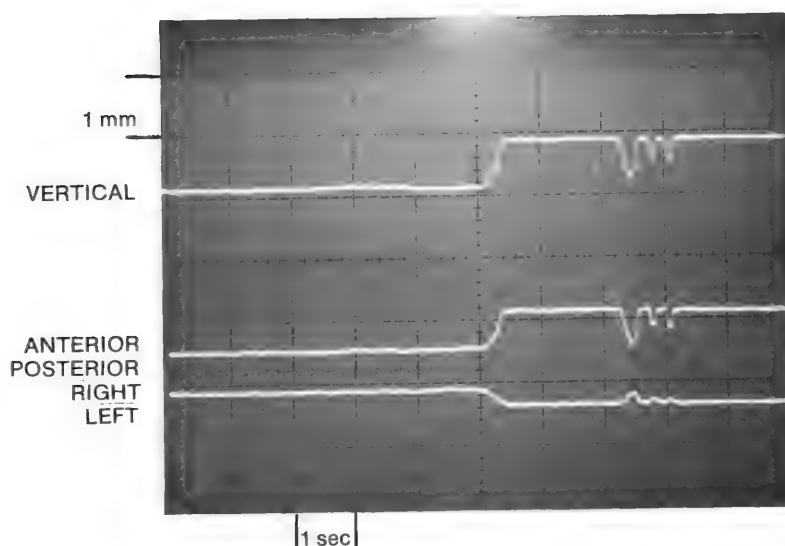
The best method currently available for measuring the mandibular position is with the Mandibular Kinesiograph.^{41, 52, 57, 84} The instrument measures

11—13. Prior to AK examination and treatment. The freeway space is only .2 mm. There is an anterior slide into centric occlusion of 1 mm and a left slide of 1 mm. See pages 319-325 for more information on interpretation of these graphs.



11—13.

11—14. After AK treatment. The freeway space is increased to .8 mm. The anterior slide is .5 mm with only .2 mm of left slide.



11—14.

Temporomandibular Joint

mandibular movement of the lower incisors in three dimensions. It is capable of recording on an oscilloscope in the X-Y axis, or by the different positions being represented by a sweep beam. Four channels of electromyography can also be combined with the mandibular movement, making this an excellent instrument to record the rest position. Further discussion of the use of the Mandibular Kinesiograph is later in this chapter.

Clinical methods for determining the rest position are used in the absence of a Mandibular Kinesiograph and electromyography. One method is to measure mandibular movement from the position of apparent rest to occlusion numerous times, and then take the average. There should be minimal variance from measurement to measurement. The method most often used is to place pieces of tape on the patient's



11—15. Measurement when patient is in the apparent rest position.



11—16. Closing to complete intercuspation reveals the interocclusal distance or rest position.

chin and nose. Measurement is taken from a point on each piece of tape when the patient is in an apparent rest position, and another measurement is taken from these points with the teeth in complete intercuspation. The difference between the two measurements is the interocclusal distance, also called freeway space. A distance of 4-5 mm, determined by the facial measurements, will reflect an interocclusal distance of 2-3 mm in the pre-molar region.⁶⁰

Ricketts¹⁰¹ has the patient say the word "BOSTON" to bring the mandible to the rest position. This gives an implosive sound from the /B/ by closing the lips, followed by an "ah" sound from the /O/ opening the lips somewhat. Next comes a fricative sound from the /S/. The /T/ gives a labiolingual sound, and finally the /N/ comes back to a neutral position. This method of bringing the patient to the rest position can be combined with the measurement of lines on tape previously described.

Another method for observing the patient in the apparent physiologic rest position is to instruct him to let his lips touch and jaws rest; the physician carefully parts the lips to observe the space between the teeth. This can be enhanced by instructing the patient to swallow, moisten his lips, and relax the lower jaw. The physician parts the lips to observe the space.²¹

If the patient is aware of the physician's intent to measure jaw position, there may be a mental override of the relaxed position. Thompson¹²⁶ recommends that the patient be engaged in conversation to distract his thoughts from himself. After speaking the individual usually swallows, and the mandible settles toward the rest position. The measurement is then taken by whatever method is being used. The physician should closely observe the patient so there is no mandibular movement prior to measurement.

A different approach for determining the distance which should be maintained between the teeth is the phonetic method described by Silverman.¹¹⁷ The patient repeats a word with the sibilant "s" in it, such as "yesss" or "Mississippi." The closest speaking distance of the teeth is located to determine the appropriate vertical dimension for planning prostheses. This is discussed more thoroughly on page 373.

Clinical methods of determining the rest position also have a reproducibility, given the same testing conditions. Comparing the two methods, the clinical method shows a resting range which is located occlusally to the EMG determined resting range in more than half the subjects studied by Garnick and Ramfjord.³⁸ It has been emphasized that reproducibility of the rest position depends on a normal

occlusion and measuring under the same postural conditions.

The rest position is used in some techniques to establish the condylar position in the mandibular fossa.⁶⁴ This method depends on balanced muscles to register the position in which the mandible should relate with the skull. This is a functionally sound principle as observed in applied kinesiology, but care must be taken that the true rest position is actually being reported.

Dombrady¹⁹ demonstrated the transient instability of the rest position with head movement, head leaning against a backrest, and with the addition of weight to the mandible. He also evaluated Silverman's¹¹⁷ theory of phonation to establish a vertical dimension and found when a patient pronounced the

word "Mississippi" loudly, as opposed to whispering the word, the dimension changed.

The complexity of determining rest position is pointed out by Ramfjord.⁹³ It is influenced by psychic tension, pain, or occlusal interferences. To this list can be added muscular imbalances resulting from postural change or muscular dysfunction. Recording the rest position in the standing position¹¹⁴ has fewer variables, such as pressure on the head distorting the skull and direct interference to the closed kinematic chain of the stomatognathic system. The standing position can create new variables if there is foot, pelvic, or other weight-bearing structural dysfunction. If weight-bearing changes are ruled out or corrected, the standing position is best for determining the rest position.

FREEWAY SPACE

Freeway space is defined as the distance between the teeth of the upper and lower arches when the jaws are in the physiologic rest position and an individual sits or stands at ease in the upright position, holding the head so that the gaze is toward the horizon.²² Normal freeway space is maintained without muscular activity, the lips lightly touching, and the tongue not placed between the arches. Freeway space is an individual consideration and varies among people.¹³² The muscles of mastication bear a relation to the other parts of the skull, making up a combination of activity not duplicated among individuals.⁷³ The relationship of the jaw to the head is established by the third month of life; thereafter it does not change.¹²⁵ This relationship is determined by the balance of the musculature and is only changed by muscular dysfunction. When normal freeway space is established for a particular person, it should not be interfered with, regardless of how great or small its dimensions.¹⁸ Shpuntoff and Shpuntoff¹¹⁴ point out the importance of maintaining normal freeway space. If this space is "... encroached upon by increasing the vertical dimension of occlusion, a constant bombardment of impulses is generated in the powerful muscles of mastication, in a tendency to restore the physiologic rest position. Unfortunately, teeth and supporting structures are often injured, and habits of clenching and grinding teeth are generated. These forces are more manifest when the patient sleeps because the restraining forces of gravity and conscious control are absent."

The importance of maintaining normal freeway space is dramatically demonstrated in the correct fitting of dentures. Jarabak⁶⁰ studied the effects of changing the freeway space by constructing three sets of dentures for individuals. One set was designed to maintain normal freeway space, one to decrease it, and one to increase it. Electromyography revealed when vertical overclosure was present, there was loss of muscle tension which frequently caused spontaneous hyperactivity. When the vertical dimension was excessive, muscle tension increased.

Before the rest position and freeway space are measured for the design of prostheses, the patient should be structurally balanced. This can be accomplished with the standard applied kinesiology methods of evaluating for and treating, if necessary, the following: temporomandibular joint function, hyoid muscular balance, cranial primary respiratory mechanism, cervical spine, head leveling, the pelvis, and the feet. The pelvis and feet are important because it is often observed that a patient has disturbance in the stomatognathic system when seated or standing, but not when recumbent. The patient who complains that a new set of dentures does not fit may very well have disturbance in some remote area. The dentures may be a perfect fit in the seated position, where the case analysis information was derived. When the patient stands there may be an entirely different set of factors present, changing the physiologic rest position and freeway space.

CENTRIC RELATION AND CENTRIC OCCLUSION

Much has been written about centric relation and centric occlusion, and there is quite a bit of controversy in the literature. A physician not in the dental field who recognizes the importance of the stomatognathic system with the total body needs to understand the relation between the condyle position and occlusion. Centric relation refers to the condyle position in the fossa, and centric occlusion refers to complete intercuspation of the teeth. Unfortunately, the neophyte in the field becomes very confused when attempting to study centric relation and centric occlusion. There is no standardized terminology, and various authors refer to the same function, position, and observation with different terms. Revealing this plethora of terms are lists found in two textbooks.^{4, 113} A combination of the lists gives some of the words used to refer to this relationship as follows: centric occlusion, centric position, centric relation, gothic arch centric relation or position, habitual rest position or occlusion, retruded centric relation, retruded rest position, terminal occlusion, terminal occlusal position, true centric relation, unstrained centric relation, centric or physiological rest position, eccentric jaw relationship, eccentric intercuspation, functional centric relation or position, normal centric relation, rest centric relation, true relaxed centric relation, true centric position, true mandibular centric position, true relaxed centric occlusion, true rest centric position, working or rest centric position. Probably the reason for the wide range of interpretations, writings, and techniques in determining centric relation is the extensive interaction of structures which influences the centric relation position.

Dawson¹⁸ considers centric relation as the starting point of occlusion, and that it is most important to the comfort, function, and health of the stomatognathic system. He defines centric relation as "... an arch-to-arch relationship of the mandible to the maxilla when both condyles are in their terminal hinge axis location *irrespective of tooth contacts*." Shore¹¹³ defines centric relation as "... a skull-to-mandible relationship. The mandible is in centric relation when the heads of the condyle exhibit pure rotary motion around the hinge-axis while the mandible traverses an arc, before transitory movement of the head of the condyle occurs." This definition takes into consideration that there is no *single* anatomical reason for the centric relationship of the mandible. The relationship of the mandible to the skull is complex; muscular balance is of primary consideration, since there is minimal restriction of joint movement by the ligaments during usual motion.⁷⁷

Centric occlusion is when the teeth of one arch have maximum occlusal contact or intercuspation with the other. There is controversy in the literature about how centric relation and centric occlusion should parallel each other. Some indicate that centric relation, defined as the most retruded superior position of the condyle in the mandibular fossa, should be the same as centric occlusion. In other words, when the condyles are forced into the most retruded superior position — the terminal hinge axis — the mandible closes into intercuspation with no "slide" into centric occlusion. Consideration of the interaction of the temporomandibular joint and occlusion with the rest of the stomatognathic system should be made to evaluate this concept.

The tremendous amount of controversy about centric relation to centric occlusion is observed by Farrar³⁴ when noting that four well-known authorities presented different views about the ideal relationship of centric occlusion to centric relation at the 1977 meeting of the American Equilibration Society. He states, "This heated debate by experienced, highly respected professionals raises a question about the significance of a precise relationship between the intercuspal position of the teeth or centric occlusion (CO) and the position of the jaw or centric relation (CR)."

This text delves into the question of centric relation only because it is often the position to which occlusion is equilibrated. As has already been noted and will be emphasized more as we continue, normal occlusion is very important to normal function of the cranium and the stomatognathic system in its entirety. Because so many health problems throughout the body originate in this system, it is mandatory that the applied kinesiologist be aware of all factors which influence it, both positively and negatively. It is not our purpose here to teach equilibration techniques; rather, it is to help the reader understand how they relate to occlusion and how centric relation relates with occlusion. To better understand centric relation, it is necessary to again consider the articular fossa, disc, and condyle relationship, along with the function of the mandibular elevator muscles. The three positions considered by various authorities as being optimum for centric relation are for the condyles to be in the mandibular fossae in the (1) most posterior superior, (2) superior, and (3) downward and forward onto the articulating eminence.⁸⁶

A common conception of the correct centric relation is with the condyle in the most posterior superior position.^{18, 49} When the condyle is in this

position, it is at the edge of joint motion.^{91, 92} There is no other joint in the body which begins its neutral position from its edge of motion position. It is obvious that this is not a physiologic neutral position, because in most normally functioning, unequilibrated individuals it can only be maintained by contraction of muscles or by passive manipulation to the location. Electromyographic studies show 76% of the subjects observed demonstrated muscle imbalance and straining when the jaws were closed with the condyles in their most retruded positions.⁸¹

The posterior area of the fossa is highly vascular and richly supplied with sensory nerve fibers; anatomically it is not structured to adequately accept forces.⁸⁶



11—17. Sectional view through left mandibular condyle and fossa of an old edentulous skull. Note thinness of the superior portion of the fossa.

Temporomandibular joint radiographs reveal that the posterior superior displacement of the condyle in its fossa is frequently associated with cases of dysfunctional temporomandibular joint arthropathy.⁴⁰ Farrar²⁸ suggests that the terms “most retruded” or “most posterior” be removed from the definition of centric relation, with the term “most superior” being sufficient. He states that there is a high percentage of iatrogenic displacement of the condyle (disc-condyle assembly) which tends to displace the disc anteriorly. It will be easier to understand this reaction to equilibrating to the most retruded superior position when the function of the disc is discussed later in this chapter.

When the occlusion is equilibrated to be in this retruded position, it is not compatible with the anterior superior pull of the masseter and internal pterygoid mandibular elevators. The superior centric relation position has this compatibility.

The condyle in the most superior aspect of the articulating fossa appears to have a more physiologic function in many ways. Here the condyle is not at the limit of joint motion. The bone of the fossa in this location is also thin; however, when the masseter and internal pterygoid contract, they pull the mandible slightly forward, which produces a slight anterior slide into centric occlusion and brings the condyle and disc into the strong bone of the articular fossa. Pressure is applied to the central portion of the disc, which is avascular and has minimal nerve supply; it is made up of dense fibrous connective tissue designed to take heavy forces without damage or painful stimulus. An important aspect of this superior position of the condyle is that it is not at the limit of the motion envelope; thus there is a range of movement for normal function.

In normal function there is a continuous change of centric relation in the process of normal adaptation.¹⁰⁴ The joint must be capable of adapting to changes in dentition^{64, 81} which it cannot do when it has to function at its edge or limit of motion. Sicher's *Oral Anatomy*²² states, “The hinge position is an extreme position, and it is entirely contrary to well-known principles of biologic constructs that any normal joint be habitually postured in such a strained position. The potential for extreme movements seems unmistakably to be an adaptation installed as a margin of safety by which the integrity of a living structure can be preserved in situations of emergency.”

Posselt⁹² favors a more relaxed position over the most retruded one for centric relation. He supports his opinion with electromyographic studies showing the posterior and middle fibers of the temporalis muscle to be highly active in the retruded position, whether it is maintained actively or passively. He further demonstrated in observations of mastication that in no case was the functional path situated on the hinge movement path or the condylar rotation path.

Silverman¹¹⁹ considers the most retruded superior position man-made, believing that the mandible does not remain in this forced position, that the condyles return immediately to their normal passive position in the mandibular fossae. He further points out that the forced manipulation of the mandible distorts the skull in reference to the mobility associated with the primary respiratory mechanism, and that the teeth may be equilibrated to this abnormal cranial balance.

The activity of the temporomandibular joint is dynamic. It is ever-changing, adapting to the needs of the time. The nervous system is constantly adapting to the current needs of mandibular action. There is a

Temporomandibular Joint

tremendous amount of variable muscle synergism available. Rapid adaptation is evidenced by the change when the teeth are lost; the habitual closing pattern seems to be obliterated rather quickly and has to be re-established after insertion of complete dentures.⁹²

Another position, considered best by some for centric relation, is an anterior inferior condylar position. This is believed to be physiological because it brings the condyle more significantly into the heavy bone of the articular eminence. A strain problem develops because this is not a balanced position for the mandibular sling. The external pterygoid must be contracted to maintain the mandible in this position.

It appears that the optimum physiologic position for centric relation is with the condyle in the superior position in the center of relaxed muscular function.^{56, 97, 110, 111, 118, 122, 124, 133} In this text this position is called "physiologic centric relation." Jankelson⁵⁶ refers to this position, as established with his Myomonitor, as myocentric occlusion. Ricketts¹⁰¹ refers to it as the alpha position to give it the status of optimum relation.

One of the reasons that centric relation in the most retruded superior position has gained so much popularity is that the position appears to be easily reproduced. It is the position used to record measurements to accurately mount study models on an articulator. Because many believe this position is reproducible, it appears to have the nicety of ease in clinical use. In reality, research by Jankelson⁵⁸ has shown that recording centric relation in the retruded position is not reproducible. In addition, the body does not operate with that simplicity. Goodheart has said from the lecture platform so many times, "The body is simply intricate and intricately simple." The normal position for centric relation is in the center of a balanced muscular system, with an adequate amount of flexibility in the position for adaptation to take place as needed by the body. The simplicity is that the mandible is suspended by the muscles at rest and in function, which automatically establishes a physiological centric relation. The intricacy is that the muscles must be functioning normally, with neither hyper- nor hypotonicity, for the balanced position to be established.

Farrar³⁵ is adamantly against equilibrating the occlusion to centric relation in the most retruded position because of its adverse influence on the disc. This is discussed further later in the chapter. For now it is adequate to observe his opinion, "The universal biological observations that nature is not precise in morphology and function also applies to the relationship between CR and CO." Moyers,⁸¹ who did considerable study of mandibular motion by

electromyography, makes a similar observation: "Most present definitions (of centric relation) fail to account for the variability always seen in biology. If there is one and only one position for centric relation in all of mankind, then it is the first trait discovered which behaves so singularly." He goes on to state, "It would seem to this writer that centric relation would have to be defined in terms of neuromuscular reflexes."

Observations of random samples of the population seem to support the ability to tolerate and function normally with a slight anterior displacement from centric relation to centric occlusion. In a study of 323 individuals, there was an anterior slide from a retruded centric relation to centric occlusion in 80% of the people studied.¹⁰² In another study of fifty seemingly normal young individuals with full complements of natural teeth, 90% could retrude their mandibles about 1 mm behind the intercuspal position.⁹¹ A hard-line philosophy that centric occlusion should parallel the most retruded superior position of the condyle and fossa seems to mean that almost everyone needs equilibration, even though there is no symptomatic disturbance or other evidence of malfunction. One school of thought⁴⁹ states that literally everyone has defective malocclusion. The objective of its proponents is to equilibrate the centric occlusion to match centric relation in its most retruded superior position. It is difficult to accept the concept that everyone has malocclusion and to also accept the concept that the body is a self-correcting, self-maintaining mechanism, which is basic to applied kinesiology. Farrar³⁵ finds an anterior deflection of up to 1 mm from posterior centric relation to centric occlusion acceptable; however, lateral deflection is unacceptable.

In determining physiologic centric relation, there is a problem similar to the one in determining the rest position and normal freeway space. The basic concept of physiologic centric relation is that the condyle-disc complex is in the median area of the articular fossa in the presence of resting muscles which are functioning normally. This means that the muscles of the mandibular sling can be neither hyper- nor hypotonic which, of course, means they must be functioning normally and not adversely influenced by remote structure. A rather long quote from the end of Ricketts' article on "Occlusion — The Medium of Dentistry"^{97*} sums up the problem of centric relation.

"The question arises as to what to register in order to treat these occlusion-joint problems.

*From Robert M. Ricketts, "Occlusion — The Medium of Dentistry," *Journal of Prosthetic Dentistry*, Vol. 21, No. 1 (January 1969), with permission.

Some will say, 'Ricketts, you do not like to register rest position for reconstruction, even though you admit to using it in rehabilitation and in treatment of occlusion and temporomandibular relations. However, you also object to using the terminal hinge axis, even though you admit it exists and can be located, and it is a point that can be found repeatedly. If you do not like either of these positions, what is left that can be registered?'

My answer is that you must always return to the musculature and to the condyle in a normal juxtaposition with the eminence through the medium of the disc or to a position of physiologic centric relation. This mandibular position is located by producing normal contraction of musculature. It is determined by the use of myophysiologic resistance to the normal closure from a position of natural upright posture. It results in a centric-condyle position away from the posterior terminal condyle location but not sufficiently forward to be on the slope of the eminence.

Any registration of occlusion is dependent upon the musculature. The muscles hold the skeletal system together; nothing else has the capacity for this stabilizing function. Whatever treatment is needed to remove contractures, to place the condyles in normal juxtaposition with the fossa, to promote the normal sensory input into the neuromuscular circuit, and to sustain functional equilibrium is the correct treatment."

Many are questioning the retruded centric relation. As a conclusion to his discussion of centric relation Matthes⁶⁸ states, "This chapter was designed to make you question what you've been taught about occlusion, especially centric relation."

With the understanding that centric relation must be a physiologic position which varies somewhat among individuals and considers the current muscular status, we go on with additional consideration of mandibular position. For centric relation to be a physiologic position, it is necessary that the muscles of the stomatognathic system function normally. The engram controlling the muscles responsible for mandibular closing is highly integrated with structural changes in the body. In normal function, the rest position of the mandible is slightly retruded in the supine position, as opposed to a seated position.⁷¹ Even though the mandible begins its closing arc from a different centric position in various postural positions, there is correct adaptation of the habitual arc to bring the teeth into unerring intercuspation. Because of the interaction of the closed kinematic chain of the stomatognathic system with remote areas of the body, it may become imbalanced under different conditions and interfere with the

engram of closing. There may be quite accurate movement of the mandible to centric occlusion when an individual is supine, but not when he sits or stands. This is usually caused from dysfunction in the weight-bearing mechanisms of the structure. There may be disturbance of the engram when upright but not when supine.

It is important that a correct engram of closing be present in all body positions. Normally this highly integrated neurologic activity is present. Because of the major interactions which can occur in different types of dysfunction, the occlusion should be evaluated in various postural positions. Physiologic centric relation and centric occlusion should be coincident, with no lateral slide from premature tooth contact in any postural position.

The dynamic action of the temporomandibular joint and muscle synergism is related by Moss,⁷⁷ who places the **general** axis of rotation of the total mandible at the site of the mandibular foramen where the inferior alveolar neurovascular bundle enters the mandible, and about which the sphenomandibular ligament attaches. This takes into consideration the dynamic forces which the numerous muscles acting in synergism have in mandibular movement. It is important to note in Moss' analysis of mandibular movement that the axis of rotation is considered general. Koski⁶³ relates to the muscular action operative on the mandible and states, "The axis cannot be located at the condyles, nor can it pass through the mandibular foramina during habitual, unstrained movements." This consideration of dynamic action of the mandible takes into consideration changes of posture, small as opposed to large opening movements, and the necessity for adaptive processes of remodeling to be able to occur. In some techniques the case plan of equilibration is developed with study models mounted on an articulator, with movement at the retruded superior hinge axis. Koski summarizes his study by stating, "Mechanical constructions based on the hypothesis of a fixed axis are unsound. The great variability of mandibular movement and the location of the axis can hardly be reproduced by a man-made mechanical device." Equilibration must be planned intra-orally in the presence of the muscular and bony framework within which mandibular movements take place.

The importance of the neuromuscular system on temporomandibular joint action should be obvious in light of the mandible's ability to go through chewing motions when the condyle is absent because of a condylectomy or a congenital anomaly.^{48, 61} To consider an exact centric relation and axis of rotation of the mandible oversimplifies a very complex and integrated action that must be able to adapt to the changing status of the body.

Form vs. Function

A question arises as to whether form determines function, or function determines form. This relates to the muscular balance, shape of the condyle and mandibular fossa, and position of the teeth. Those who deal primarily with equilibration attempt to eliminate any prematurities which might stimulate the periodontal ligament proprioceptors to influence muscular action. It is well-known that numerous obnoxious contacts in an occlusion create confusion within the nervous system in an attempt to find a muscular balance to eliminate the malocclusion. Eliminating the prematurities by selective grinding is a classic example of form determining function.

Function determining form is evaluating for and correcting muscular and other imbalances to allow the teeth and TMJ to adapt to correct balance by natural remodeling processes. An example of muscular imbalance affecting occlusion is an anterior tongue thrust against the upper central incisors. Continued thrusting action pushes the teeth into labioversion. Re-training the tongue to contact the incisive papilla during swallowing takes the pressure off the teeth and, if the lips have normal strength, the teeth tend to return to normal position. There may not be enough natural remodeling of the alveolar bone to completely return the teeth to their normal position, and an orthodontic appliance may be needed. The major consideration here is that function of the tongue may determine the form of the teeth. Return to normal muscular function is necessary, even if orthodontic procedures are used. Failure to do so may cause a relapse to abnormal form as soon as the orthodontic appliances and retainers are removed.

There are proponents of both form determining function and function determining form. Either is possible, and the answer to the question is found by determining the etiology of the malocclusion so that the correct therapeutic regime may be designed and instituted. There are times when the primary etiology is cranial faults which change the position of the dentition; the dentition may have changed as the result of appliances or restorations. When the occlusion changes and thus affects the mandibular position, causing temporomandibular joint disturbance, the cause of the malocclusion must be corrected. The muscles of mastication or the orofacial muscles may be at fault. Direct treatment to the muscles or exercises to develop muscular balance, such as myofunctional therapy, may be necessary. If the disturbance is due to a restoration, equilibration is the treatment of choice. When the etiology of the problem is diagnosed and proper treatment is administered, the temporomandibular

joint returns to function in its physiologic centric relation.

Disturbed Centric Relation

To understand physiologic centric relation, it is important to have firmly in mind the many ways that the temporomandibular joint may be disturbed and thus affect centric relation. As previously stated, the muscles of mastication can be influenced by remote factors, as well as by the engram which develops from the way the occlusion stimulates the periodontal proprioceptors. The muscles may be imbalanced as a result of cranial faults, the correction of which may change the mandibular relation to the skull. The muscles of mastication are variably influenced by the closed kinematic chain of the stomatognathic system,³⁷ which in turn is influenced by body posture throughout. Muscles in the chain are also influenced by stimulation of remote proprioceptors, such as in the feet and pelvis. Centric relation is dynamic. It is different when one is tense and tired as opposed to refreshed and relaxed, or afraid as opposed to quiet and at ease. Centric relation is a neurologic concept. The reflexes controlling centric relation must have been learned and be capable of some learning.⁸¹ This more dynamic consideration of centric relation takes into consideration the fact that muscles influence structure and that the TMJ itself is loose, with no great restraint by the ligaments.⁷⁷

Again, it is not the purpose of this discussion to indicate how equilibration should be accomplished; rather, we want to consider the many variables present in normal TMJ function. There is considerable evidence that a rigid interpretation of centric occlusion matching centric relation, defined as the most retruded superior position, is problematic, to say the least.²²

Remodeling of the temporomandibular joint takes place throughout adult life, leading to marked and typical changes in shape. Mongini⁷⁵ states, "These findings cast doubt on the proposition that the gnathological determinants (particularly centric relation) are never changed in the course of time." Ricketts¹⁰¹ does not believe the TMJ remodels except in the presence of pathologic stress or disease processes. The degree of TMJ remodeling is closely related with the dentition. This constant adaptation between the TMJ and the dentition enables the two to function harmoniously. The ongoing remodeling is capable of maintaining this harmony, except when the change in muscular action or occlusion is rapid and cannot be accommodated with natural processes. This may occur as a result of tooth restorations or extractions, traumatic cranial faults,

or injury to the involved muscles. The change of occlusion from dental restorations and its effect on the TMJ are obvious. This type of occlusal disharmony with physiologic centric relation is purely a dental problem which should be corrected by equilibration. The muscular and cranial primary respiratory factors influencing centric relation require additional discussion.

Cranial Faults

There are many ways cranial faults can cause malocclusion. One is by actual change of the cranial bone position. The two mandibular fossae have generally been considered as rigid components with each other, thus providing a solid base with which the mandible can relate. In reality, there is a physiological motion of the temporal bones which causes the mandibular fossae to be independent of each other. The normal physiological motion of a temporal bone is a mirror image of the contralateral temporal bone; thus the mandibular fossae move synchronously with each other. During spheno-basilar inspiration or expiration, the mandibular fossae of the temporal bones rock slightly but parallel with each other to not greatly influence the occlusion. This balanced activity is necessary for normal function of the temporomandibular joint and occlusion. In certain types of cranial faults, there is an internal rotation of one temporal bone and an external rotation of the other; thus the mandibular fossae are imbalanced, causing displacement of the mandible. If an individual is critically equilibrated to centric occlusion in the most retruded superior centric relation position with these cranial faults present, the occlusion will be made to match an abnormal skull. The cranial faults probably cannot be corrected under these circumstances because every time an individual bites down, the powerful muscles of mastication make even a recently corrected skull match the occlusion, and the cranial fault is perpetuated. Henningsen,⁴⁷ a dentist, recommends observing for a lateral shift during mandibular opening as a clue to misalignment of the temporal bone. The lateral shift could also be due to muscular imbalance.

Muscular Imbalance

Muscular imbalance can adversely influence the mandibular position, consequently changing the physiologic centric relation. Mandibular muscles are normally controlled by the engram developed from stimulation of the periodontal ligament proprioceptors. The muscles must be capable of responding correctly to the engram. In some cases, a muscle can be injured so that its proprioceptors interfere with the normal pattern. This can happen as a result of any activity which stretches or contracts the muscle

past its physiological ability to respond. An example is dental procedures which require a patient's mouth to be forced open for prolonged periods. This type of muscular imbalance can be readily diagnosed and treated with applied kinesiology procedures, described in Chapter 14. Muscular imbalance can result from the engram being disturbed by improper stimulation of the periodontal proprioceptors, as described in Chapter 9. The muscles can also be out of balance as a result of cranial faults which are causing peripheral entrapment of cranial nerve V. In this case a combination of cranial faults and muscular imbalance is probably influencing centric relation. Muscular imbalance for any reason may cause the mandible to close in such a manner that prematurity develops. If an individual is equilibrated with muscular imbalance present, centric occlusion will be made to match an abnormal condition.

When studying centric relation, centric occlusion, rest position, and the resultant freeway space, it is important to consider all factors which influence these positions. There should be no argument as to whether function dictates form or form dictates function; both are applicable. In making a case study, the primary cause of dysfunction must be determined. If malocclusion is a result of new restorations or other change in the occlusion, equilibration is necessary to return normality. If, on the other hand, the malocclusion is caused by muscular imbalance, its etiology must be determined. Is there disturbance in the periodontal ligaments, abnormally stimulating the proprioceptors to change the engram of muscle function? If so, local treatment to a tooth may be indicated and may be as simple as manipulation, described in Chapter 9. If the muscle proprioceptors are dysfunctioning from injury to the muscle, they should be evaluated and treated. If centric occlusion and centric relation are not in harmony as a result of cranial faults changing the position of centric relation, then the cranial faults should be corrected. In none of these cases is selective grinding for equilibration indicated; in fact, it may cause the perpetuation of health problems or create additional ones. Before determining that selective grinding is necessary, the question should be asked, "Why is it necessary?" The body is a self-perpetuating, self-maintaining mechanism. The teeth do not come out of occlusal harmony for no reason. Is the grinding — an irreversible change — necessary, or is some other factor of TMJ function causing the malocclusion? Only after the temporomandibular joint and its muscles, along with the total stomatognathic complex and remote factors such as spinal and pelvic subluxations, feet, and general structural and muscular balance, are evaluated should selective occlusal grinding be done.

TMJ and Mandibular Movement

As we progress into the movement of the temporomandibular joint, we find the controversy about the TMJ continuing. The literature describes the motion of the temporomandibular joint in various ways. Again, the numerous descriptions are probably because of the complexity of this joint and the large amount of synergistic activity which must take place during its movement. It is of value to again look at Koski's⁶³ comment on mandibular motion: "It may not be easy to prove where the axis of the opening movement of the mandible is located, but it appears quite easy to show where it *cannot* be. The axis cannot be located at the condyles, nor can it pass through the mandibular foramina during habitual, unstrained movements." As we will see later in this chapter, when measurement of mandibular motion is considered there is no constant axis of rotation. The rotation of the mandible varies, depending on the activity of the muscles under neurologic control. It varies from position to position, depending on the individual's posture and other factors in the stomatognathic system, and possibly from remote influence from numerous areas of the body.

Motion available from the temporomandibular joint is that of mandibular opening and closing, protrusion and retrusion, and lateral motion. The TMJ is classified as a ginglymus and gliding joint.⁴⁵ The combination enables this unique joint to act in various combinations to provide the movement necessary for complex activities, such as trituration; it also allows the mandibular opening to adapt to man's upright position, with the neck running vertically downward in close proximity to the posterior aspect of the mandible.²⁰

The uniqueness of the temporomandibular joint is due to several factors. As pointed out in Sicher's *Oral Anatomy*,²² the articulation of the mandible with the skull can be considered as one individual activity, since it is impossible to move one articulation without influencing the other. In some activities one joint acts more as a stabilizing force, while the other makes the mass movement influencing mandibular position. In some motions, such as straight opening and closing, the joints move symmetrically as they may in protrusive and retrusive movements. The disc and its two compartments are necessary for allowing the mismatched mandibular fossa and condyle to function together. The two compartments above and below the disc add to the motion capabilities.

Movement of the mandible is classified as bilaterally symmetrical or bilaterally asymmetrical. The four symmetrical movements are protrusion, retrusion, depression, and elevation. The asymmetrical movements are laterally left and right. Most TMJ motions require a great degree of muscle

synergism. Possibly the most important factor about the movement of these joints is that they are the only joints of the body having teeth, giving a unique proprioceptive action to the muscles' synergism. The uniqueness of the complex is emphasized by the muscle harmony and action when the joint is incomplete. An individual who has lost the condyle from surgery or congenital anomaly can move the mandible through most of its range of motion by *muscle action alone*.^{48, 61} Although there is some guiding of mandibular motion by the joint surfaces, motion is directed more by the musculature and less by the shape of the articulating bones and the articular ligaments in the temporomandibular joint articulation than in other joints. Dysfunction is not caused by lax ligaments, but by disturbance in the intricate neuromuscular mechanisms controlling mandibular movement.¹⁰⁷ This is not to say that pressure is not exerted into the joint. As Moss⁷⁷ points out, "... pressure as in all diarthroses is *always* present in the temporomandibular joint during function. It can be said that where there is not pressure there is no function."

The uniqueness of this complex is further emphasized because it is the only place in the body where there is a muscular complex attached at only one end. The tongue, with all its intricate movements, is coordinated with jaw activity; rarely does it ever get between the teeth in normal function and get bitten. The tongue is included in the orofacial muscle group. When these muscles are functioning improperly in swallowing and in their other actions, they contribute to temporomandibular joint dysfunction.¹²⁰ Applied kinesiology has some approaches to correcting dysfunction of the orofacial muscles. Myofunctional therapy, discussed in Chapter 15, may be necessary.

Movements of the mandible can be classified into three types of function. (1) There is free movement, where there is no occlusion and the mouth is empty. This occurs during speaking, yawning, coughing, etc. Neurologically it relates with muscle and joint proprioceptors. (2) Masticatory movement is involved with chewing and includes all types of movements. The muscles are controlled neurologically by stimulation of the periodontal ligament proprioceptors and sensory feedback from the oromucous membranes, as well as from the joint and muscle proprioceptors. (3) There are mandibular reflex movements which are jaw-opening, jaw-closing, and jaw-jerk reflexes. The jaw-opening reflex is from stimulation of the periodontal ligament proprioceptors; it is discussed in Chapter 9. The jaw-closing reflex relates with swallowing and is discussed in Chapter 10. The jaw-jerk reflex is a myotatic reflex of the jaw-closing muscles.

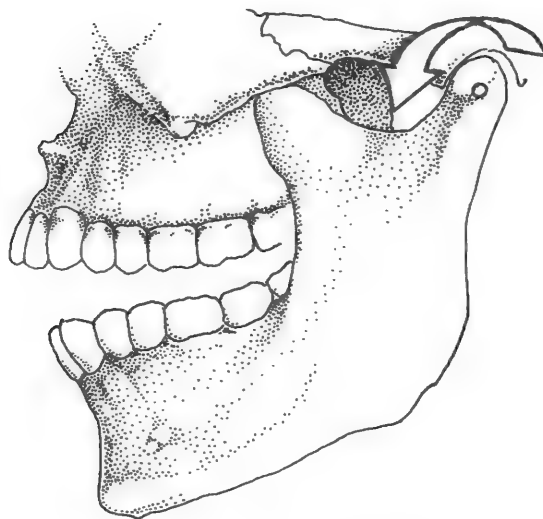
ROTATION AND TRANSLATION

The motions of the temporomandibular joint are rotation and translation. The term "rotation" refers to a body rotating about one of its main axes; it is measured in degrees of an angle. "Translation" refers to displacing the total structure from one point in space to another, exhibiting a change in total position. Translatory movement is measured in linear units. Rotation in the temporomandibular joint refers to movement of the condyle around its central axis, and translation refers to the sliding of the condyle on the articular eminence. Most mandibular movements are a combination of both. Rotation — a hinge-like action — can only be accomplished during the first stage of mandibular opening as an individual action. The terminal hinge position is "... the posture in which the condyles rest at the most retruded limit, against the thick back rims of the discs below the front of the fossa when the cusps of the teeth are just cleared of contact."²² This position is claimed to be located with accuracy and is the position when "pure" hinge raising and lowering of the jaw can be done. Most often the terminal hinge position is considered to be centric relation. Some feel that centric occlusion should be identical to that position. We have already considered that *Sicher's Oral Anatomy*²² points out that the hinge position is

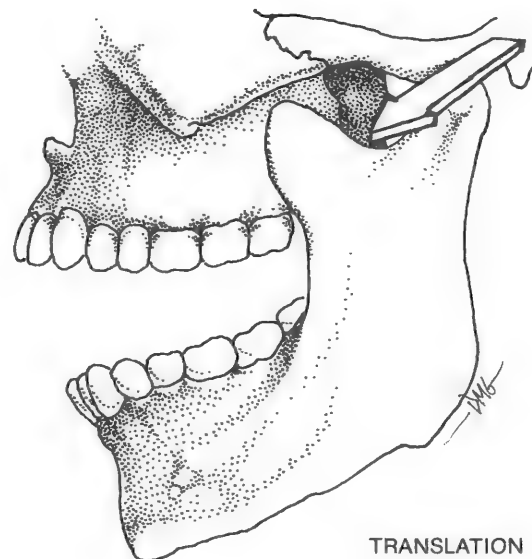
extreme and states that "... it is entirely contrary to well-known principles of biologic constructs that any normal joint be habitually postured in such a strained position. The potential for extreme movements seems unmistakably to be an adaptation installed as a margin of safety by which the integrity of living structure can be preserved in situations of emergency." Applied kinesiology observations of TMJ function, described in Chapter 14, agree with this.

It is generally agreed that rotation occurs in the lower compartment between the articular disc and the head of the condyle; thus the condyle moves in relation to the meniscus. The action of translation occurs in the upper compartment where the disc slides on the articular eminence.

The external pterygoid muscle consists of a superior and an inferior head.⁴⁴ The larger inferior head acts in opening movements to pull the mandible and articular disc forward, thus maintaining the disc and condyle as a functional unit. The superior head is active during closing movements of chewing or clenching the teeth, and during deglutition to stabilize the condylar head and disc against the articular eminence during closing movements of the mandible.⁷²



ROTATION



TRANSLATION

11—18. Temporomandibular joint motion is most often a combination of rotation and translation.

Temporomandibular Joint

Posselt's Envelope of Motion

Posselt^{91, 92} described an envelope of motion of the mandible in the sagittal and horizontal planes. The research was done by attaching a stylus to the mandibular arch by means of clutches which did not interfere with the usual occlusion. The stylus scribed the various arcs of motion onto a fixed plate. Posselt's original work has been verified by various electronic devices; it is generally accepted as the motion of the mandible. The outer borders of the envelope as illustrated in figures 11—19, 11—21, and 11—22 are the extremes of range of motion. The dimensions of this motion are limited by the ligaments, the articulation, tooth contact, and possibly by muscle shortness.

Terminal hinge movement. The terminal hinge movement is the mandibular movement available when the condyle is in the most retruded superior position. This is a stable position, so movement is of a pure hinge nature. This motion (illustrated by I-II in figure 11—19) is sometimes called the centric relation arc. The maximum amount of movement in this arc separates the upper and lower incisors from 20-25 mm. It is limited to the motion obtained while the condyle remains in the most retruded superior position of the fossa.

Movement through the terminal hinge arc can be accomplished actively by the subject contracting the middle and posterior fibers of the temporalis muscle to maintain the posterior position of the condyle; the external pterygoid muscle is kept relaxed to avoid translation. Movement through this arc can also be passive, with the subject maintaining relaxation in the jaw muscles.

Motion takes place in the terminal hinge arc in the lower joint compartment, with the condyles rotating on the disc while it remains stationary. Motion past the limit of the terminal hinge arc (II) requires translation to begin.

There is considerable controversy regarding the practical use of the terminal hinge arc. It appears to be reproducible because the condyles are forced into their most posterior superior position, producing a pure hinge action. This position is said to record a stable centric relation to accurately mount study models in an articulator. Some consider that centric occlusion should be on this arc, and equilibration is done to accomplish this. Others disagree, considering that this is a maximum border motion and the articulation must be in stress in this forced position. In support of this, Posselt⁹² states, "... electromyographic registrations indicate that to maintain a terminal hinge (retruded) position the posterior and middle fibers of the temporal muscle are highly active. This is the case whether the position is taken

up actively or passively or whether retrusion is achieved along a flat or oblique plane." He continues to support the concept that ideal centric occlusion is within the extremes of outer border movement and on the habitual path of closure. He states, "Support for the concept of the (ideal) intra-border type of (centric) area lies in the fact that about 90% of fifty seemingly normal young individuals with full complements of natural teeth can retrude their mandibles about 1 mm behind the intercuspal position."

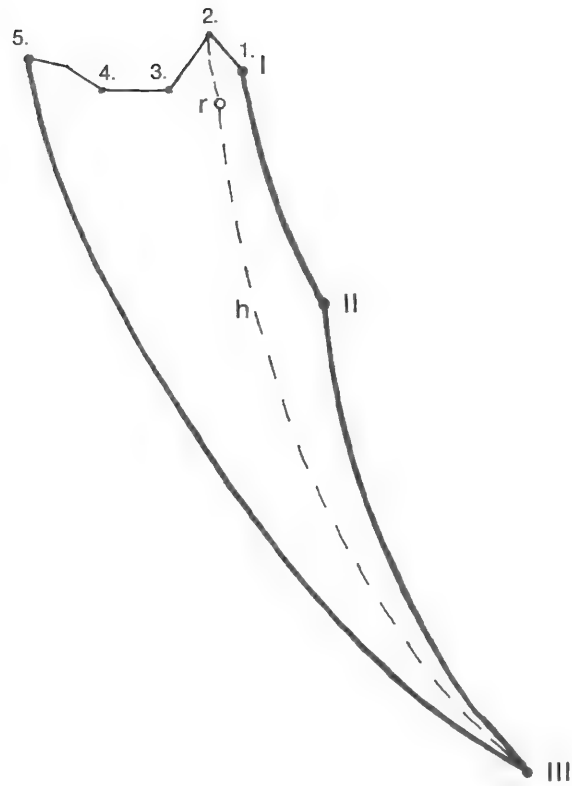
Translation arc. The translation arc (II-III) continues the maximum border movement of the envelope, beginning at the limit of the terminal hinge arc. At this point it is necessary for translation to begin if opening is to continue; thus the condyles translate inferiorly and anteriorly over the articular eminence. This motion is of the upper compartment, with the disc sliding on the articular eminence. The lower compartment maintains its relation with the condyle, moving as a unit. An additional 30-35 mm of opening between the incisors is available from this action, making the total maximum opening in adults 50-60 mm. This is the total opening; it allows for vertical overlap of the incisor teeth which may exist prior to movement (see page 255 for discussion of maximum opening).

It is on this arc that disc movement in relation to the condyle may develop, causing popping and cracking of the jaw. As long as motion takes place in the terminal hinge arc, there is no shift of the disc, as will be discussed later in this chapter.

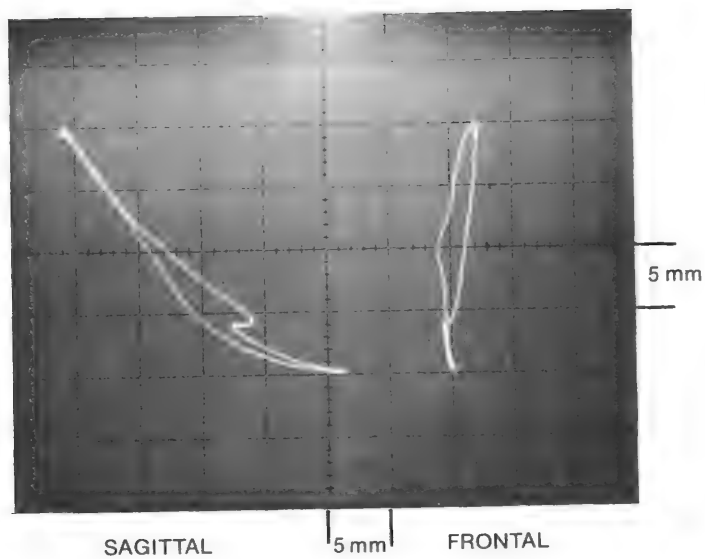
Occlusal limitations. The top portion of Posselt's envelope begins at (1), which is the most retruded superior condylar position in the articular fossa. Centric occlusion is located at (2) where there is maximum intercuspation. The edge-to-edge position of the central incisors is at (3); (4-5) is movement from the edge-to-edge contact of the central incisors to mandibular protrusion with a reversed vertical overlap; and (5) is the protruded contact position.

Anterior border movement: This movement (5-III) is opening or closing from the protruded contact position to the maximal translatory opening while the mandible is maintained in maximum protrusion. In closing along the anterior border, the condyle loses some of its maximum translation on the articular eminence as the mandible moves toward the protruded contact position of the central incisors. This movement is limited by the ligaments and muscles; it is only of academic interest and has slight practical value.

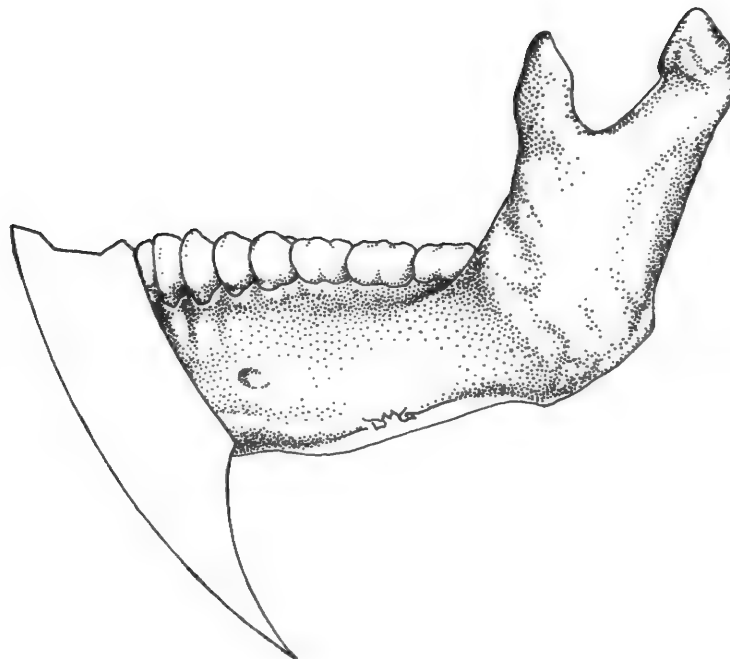
Habitual arc. The habitual (h) arc is traced by the dotted line from maximum opening (III) to (2) centric occlusion. This movement takes place within the confines of the border movements described



11—19. Posselt's envelope of motion.



11—20. Trace on Mandibular Kinesiograph of patient with TMJ dysfunction. Note the failure of the opening and closing traces to parallel each other. The notch on opening is disc dysfunction producing an "opening click."



11—21. *Lateral representation of Posselt's envelope.*

above. It is the path an individual would take from maximum opening to centric occlusion, or from centric occlusion to maximum opening. Repeated habitual opening and closing movements do not coincide exactly, but they have a fairly characteristic main course. It is governed by the proprioceptive system being a conditioned reflex which appears to become more accurate as it moves toward centric occlusion. This is due to the proprioceptive periodontal ligaments which guide the muscles to come quickly and unerringly to centric occlusion. The exactness of this guidance system can be observed in the rapidity with which the engram changes when a prematurity develops. A new muscular closing pattern develops to avoid the prematurity, if structurally possible.

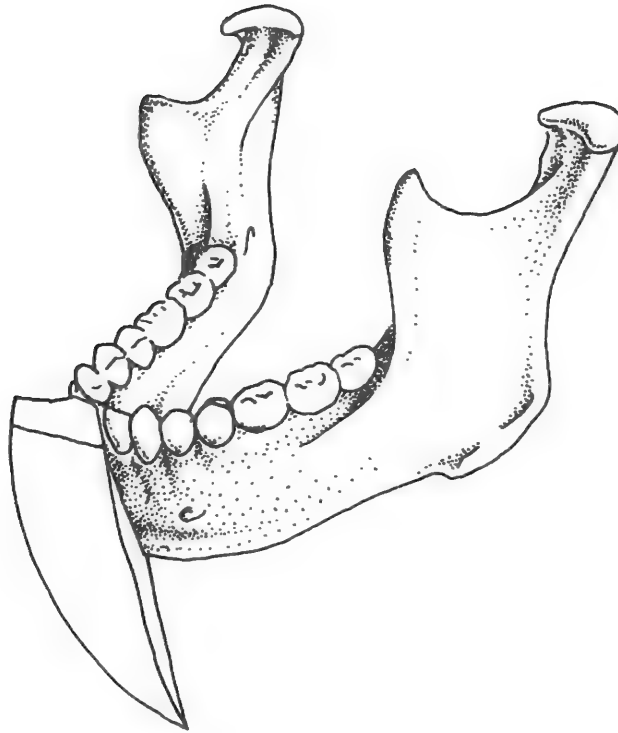
The habitual arc will vary with changes in the closed kinematic chain of the stomatognathic system. The variable may be a normal postural change, such as flexion or extension of the cervical spine, or it might be abnormal, such as imbalance of the muscles of the hyoid. It could also be remote influence from proprioceptors in the feet, pelvis, etc., sending aberrant information to the stomatognathic system.

In the basic area of the habitual closing arc, relatively close to centric occlusion, is the habitual rest position (r). As has been discussed, this is

usually a reproducible position which is important in establishing the vertical dimension of occlusion.

Lateral movements. Movement of the mandible in the transverse plane (horizontal) has been plotted by Posselt,^{91, 92} starting with the condyles in the posterior superior position. This is the retruded centric relation position, often defined as the most posterior position from which the mandible can perform lateral movements. Posselt⁹² points out that there is no more posterior position than this; consequently, the definition is superfluous.

Tracing of lateral movement from the most retruded position results in an angular tracing called the "gothic arch" or "arrow point" tracing. If a stylus is attached to the mandible, the apex of the tracing points posteriorly. The gothic arch tracing can be registered actively or passively; it is practically identical in either procedure. To form the gothic arch tracing, a subject moves the mandible laterally and anteriorly from the most retruded position in both directions. A limit of lateral motion will be delineated. Further protrusive motion from this position requires the jaw to return to the median line. Continued tracing in this manner will form a rhomboid figure of the border of lateral movement combined with retrusion and protrusion. This recording, made at different degrees of vertical opening, can be combined with the sagittal tracing to provide a three-



11—22. Three-dimensional representation of Posselt's envelope.

dimensional recording of mandibular movement (figure 11—22). Note that at the inferior border, movement is limited to a small point, with minimal lateral protrusive or retrusive movements.

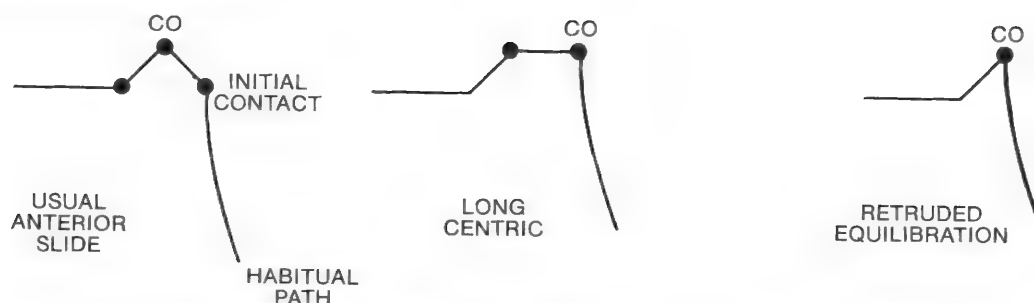
With an understanding of Posselt's envelope of motion, the previous discussion of the optimum centric relation can be graphically displayed. There are two arcs of motion in mandibular closing. One is the terminal hinge position which is at the limit of the motion envelope. In most individuals who have not been equilibrated, the uppermost point of this arc brings the teeth together but not in complete intercuspation. An anterior and superior slide brings the teeth into centric occlusion in these individuals.^{35, 49, 92} This terminal hinge closing is done only under conscious contraction of muscles to hold the terminal hinge position on an active basis, or it is held in that position on a passive basis by the physician. The normal closing pattern is the habitual one. This is directed by the engram which controls the muscles of closing; it is developed by proprioceptive stimulation. The proprioceptors that build this engram are located in the periodontal ligament, gingiva, mucous membranes, and ligaments and tissues of the TMJ itself.⁶¹ This is the path of closing when an individual consciously snaps the jaws closed to unerringly move

into centric occlusion. It is also the pathway when the jaws close from rest position to centric occlusion or during swallowing, chewing, and other activities which cause the jaws to close.

Some consider the difference between closure at the terminal hinge position and the deflection anterior and superior to centric occlusion to be deflective malocclusion,^{18, 49} while others consider the anterior superior slide to be physiologic, acceptable, and even necessary.^{35, 92, 97}

There are two concepts in equilibration theory to eliminate what is considered a deflective malocclusion from the terminal hinge position to centric occlusion. The gnathologic concept uses selective grinding to equilibrate the occlusion, making the terminal hinge position and centric occlusion coincident. Thus there is one pathway of closure, since the terminal hinge position and the neurologically guided habitual arc will terminate in the same centric occlusion. This causes the temporomandibular joint to function in the most retruded superior position, which is the edge of motion graphically shown by Posselt's envelope of motion. This does not put centric relation in the center of balanced muscle function as is seen in normally functioning unequilibrated individuals.

Temporomandibular Joint



11—23. Drawings are of different types of occlusion at centric occlusion of Posselt's envelope.

The second concept of equilibration is called long centric. Here the teeth are selectively ground to put the terminal hinge position and centric occlusion on the same vertical dimension. This permits an anterior slide to centric occlusion with no superior slide from the terminal hinge position.

There is controversy as to whether an anterior superior slide of the mandible from the terminal hinge position to centric occlusion is acceptable. From the applied kinesiology viewpoint, it is not only acceptable but desirable, because it puts the joint in the center of muscular balance for its function. The terminal hinge position is unphysiologic because it is at the edge of available motion. The neutral centric position also seems to be best because the anterior superior pull of the masseter and internal pterygoid tends to move the condyle and disc somewhat anteriorly to buttress against the articular eminence, a stronger section of the fossa. It seems to be generally agreed that a lateral slide to centric occlusion from either the terminal hinge position or the habitual arc is undesirable and detrimental to the teeth involved.

As we have progressed with our temporomandibular joint discussion, there has been much reference to the engram that is developed as a result of proprioceptive stimulation of the periodontal ligament, gingiva, oromucous membranes, and the joint itself. This system of proprioceptors must be (1) functioning normally at the receptor, (2) interpreted correctly in the central nervous system, and (3) capable of controlling the muscles properly to have a consistently correct closure on the habitual arc. It serves here only to reiterate how disturbance can develop so that a balanced physiologic centric occlusion is not operative. As previously noted, a simple prematurity is managed by the nervous system; a new engram is built which avoids the prematurity and brings back a non-stressful occlusion. This can happen only if the prematurity is within the normal range of function. If numerous prematureties conflict so that the nervous system cannot provide an habitual arc which closes the jaws without

prematurity, malocclusion will be present with its resultant stress. Munro⁸³ graphically demonstrated with electromyography the confusion which develops within the muscular system of the temporomandibular joint when prematureties are present. He demonstrated activity of the mandibular closing muscles while the mandible was opening. Obviously antagonistic muscles functioning at the same time are stressful activity and certainly not functional. The same type of disturbance can result from a neurologic tooth (Chapter 9) observed in applied kinesiology. If there is improper stimulation of the periodontal ligament proprioceptors, for whatever reason, the pattern of the habitual arc — as well as physiologic centric relation — will change. The pathway of the afferent supply of the mucous membrane, joint, teeth, and muscles through cranial nerve V to the mesencephalic and trigeminal nuclei and the efferent pathway back to the muscles^{13, 14} reveals possible disturbance as a result of cranial faults. Finally, it is necessary for the muscle proprioceptors not only to send proper afferent information, but to be able to respond to the efferent information. If there is disturbance in these proprioceptors, as is sometimes observed with applied kinesiology techniques, the muscles themselves will cause an improper closing pattern on the habitual arc, changing the physiologic centric relation. The applied kinesiology system of evaluating the muscles for balance and function is described in Chapter 14.

Lack of harmony in muscle function, which is responsible for an improper habitual arc, relates with hypertonicity in some muscles, weakness in others, and probably general stress in all. Hypertonicity or spasm is blamed for much of the pain in temporomandibular joint dysfunction.¹⁰⁵ This seems to be true whether the pain is sharp and localized, or dull and diffuse. Trigger points affecting temporomandibular joint dysfunction as described by Travell¹²⁷ are generally located in the muscles. They may be located within a muscle which is hypertonic, or may be causing the problem from a remote muscle.

Methods of Measuring Mandibular and TMJ Motion

Temporomandibular joint motion is generally not measured on a clinical basis; rather, it is observed. The physician palpates the temporomandibular joints as the mandible is put through its range of motion. Palpation is for crepitation, popping, smooth movement, and abnormal movements of the discs. Joint position can be observed by palpating the posterior aspect of the temporomandibular joint through the external auditory canal. The mandible is also observed for lateral deviation on opening, and for other aberrant movements through its entire range of motion. Ideally, an objective measurement of temporomandibular joint movement should be available, especially for research.

There are many methods of measuring TMJ motion which are used primarily on a research basis. Some are applicable to clinical use for those specializing in temporomandibular joint function. A brief description will be given here to acquaint those who wish to proceed with research or improve clinical ability with the equipment and procedures.

Mechanical Methods

Most of the early measurement of mandibular movement was done by attaching mechanical devices to the jaw, usually by way of the teeth. The attachment device to the teeth is called a clutch. The problem with this method is that any foreign object attached to the mandible may interfere with normal function. The pressure applied to the teeth can stimulate the periodontal proprioceptors, influencing neurologic control of mandibular movement. Foreign objects attached to the jaw also add weight that is potentially significant enough to change mandibular function. Some of the devices which have been used even interfere with complete intercuspation of the teeth.

Devices attached to the jaw by clutches or mandibular plates have various principles for recording movement. The pantograph is a system of scribing motion on an internal or external writing surface. Telemetric devices send radio signals to be received and recorded. The primary considerations are the weight of the device and method of attachment. There is the potential of influencing mandibular movement so that the subject's usual action is not truly being recorded. Usually a considerable amount of preparation is needed to make clutches or plates which attach the devices to the patients; this precludes testing large groups of individuals in an efficient and economical manner.

X-Ray and Photographic

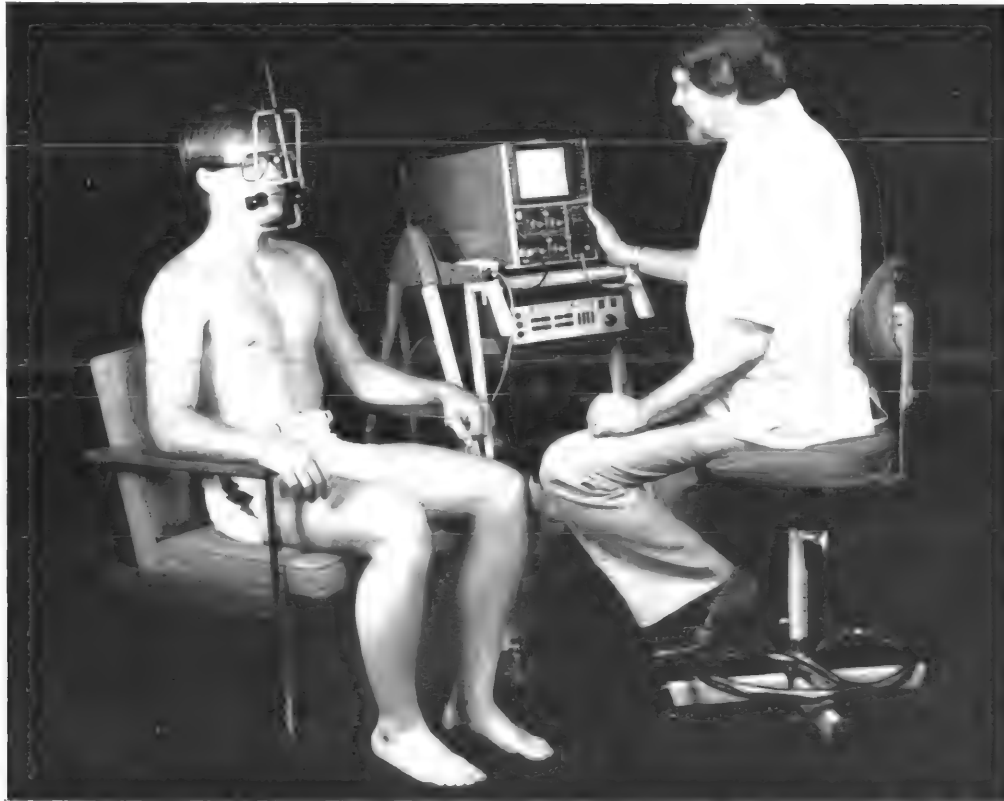
Cineroentgenography has added considerable information about the movement of the mandible and its interrelationship with the total stomatognathic system.⁵¹ Although excellent information can be derived from this system, it has the drawbacks of radiation to the individual and a limited number of evaluations of the movement to determine repeatability. There is also an inability to frequently re-evaluate an individual following therapeutic efforts; other major drawbacks are cost and limitation of equipment available for clinical use.

A relatively simple procedure of recording mandibular motion is described by Dombrady.¹⁹ Mandibular motion is recorded by attaching a small electric light bulb to the lower jaw, which exposes film in a camera. The procedure is done in a darkened room. In Dombrady's set-up the camera was set on a turntable which was moved slowly by a clock-like mechanism so that the moving light recorded a sweep of the mandibular motion across the film. An additional light was placed on the forehead as a control against head movement. Still another light was attached above the forehead lamp and controlled by an interrupting circuit recording a time interval. Recording motion in the sagittal jaw opening-closing position will document lateral deviations caused from lack of disc harmony with the condyle or from muscular imbalances.

Mandibular Kinesiograph

Jankelson et al.⁵² have developed an instrument to measure mandibular motion on an oscilloscope. The instrument — the Mandibular Kinesiograph (MKG) — relates the spatial location of a permanent magnet attached to the mandibular incisors. The sensing device is an array of magnetometers located peripherally to the orofacial area. The recording displays the three-dimensional position of the mandible through various movements. It can trace the mandible's activity from centric relation to centric occlusion, through its range of motion, and back to intercuspation. It is used to locate the rest position, determine if the muscles of mastication are relaxed, and observe motion during chewing. It is also used to document prematurities, jaw-clicking, and associated disc involvement. The instrument can be used for post-treatment evaluation, with permanent records of the oscilloscope tracing kept on photographs.

The mandible is traced where the magnet is attached to or below the lower central incisors. The



11—24. Mandibular Kinesiograph.

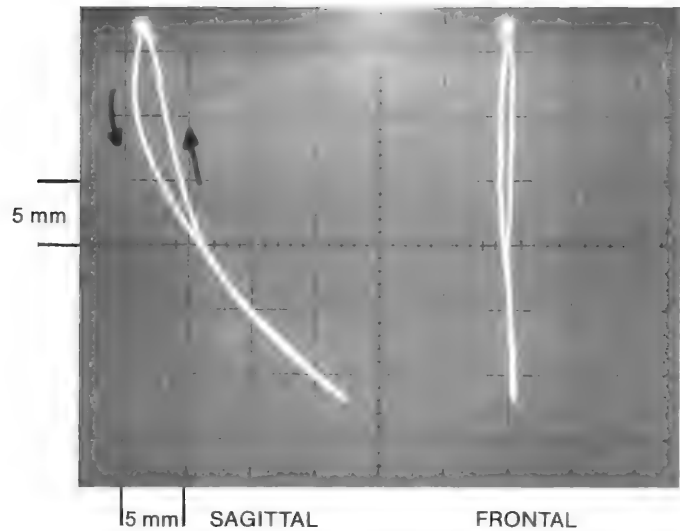
three-dimensional motion of the mandible can be traced in the X-Y mode (figure 11—26), or the traces can be made to sweep across the oscilloscope (figure 11—27).

Figure 11—26 shows relaxed opening and closing in the habitual arc. The left tracing demonstrates vertical and anterior-posterior movement, and the right, lateral movement. The ideal movement has no lateral deviation; there is a smooth curve from the sagittal view, with the closing arc overlying the opening arc. In figure 11—26 the mandible deviates 1 mm to the left opening, and closes in a straight line as seen in the right tracing. Viewed sagittally, the mandible moves anteriorly out of intercuspation, indicating that centric occlusion and the condyles may be slightly retruded in relation to the current neuromuscular balance which establishes the habitual opening arc. In this motion — which was not forced — there was approximately 30 mm of opening. Mandibular motion indicated on this graph is within the normal range. Applied kinesiology examination did not reveal any dysfunction.

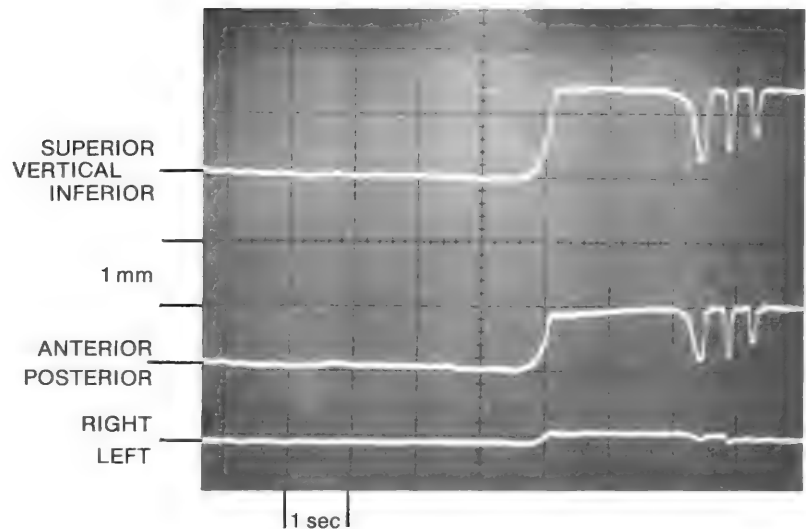


11—25. Close-up of sensor array.

11—26. Relaxed opening and closing. See text.



11—27. First half of graph is the mandibular rest position. The patient then closes into intercuspation and taps the teeth together three times. See text.



Figures 11—27, 11—28, and 11—29 illustrate the three-dimensional movement of the mandible in the sweep mode which produces horizontal traces. This evaluation is excellent for measuring rest position stability, amount of freeway space, and deflections of the mandible upon entering or leaving intercuspation.

While in the sweep mode, the deflection from prematurity can be easily measured. The movements in graph 11—27 are (1) top trace: vertical movement in the sagittal plane, (2) middle trace: anterior-posterior movement in the sagittal plane, and (3) bottom trace: lateral movement in the transverse plane.

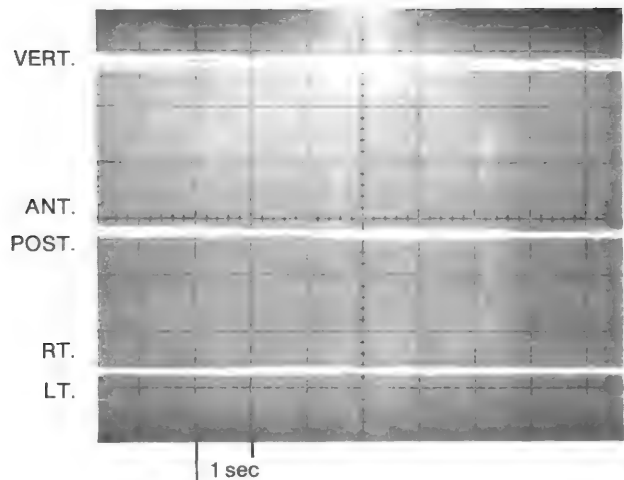
The first half of graph 11—27 represents the mandible's stable rest position. At approximately the mid portion of the graph the subject is requested to close the teeth. Movement of the trace at the top reveals 1.3 mm of freeway space. The middle and lower

traces show .8 mm of anterior slide and .2 mm of right lateral slide into centric occlusion. Ideally there is no lateral slide into centric occlusion, but this graph is within normal limits.

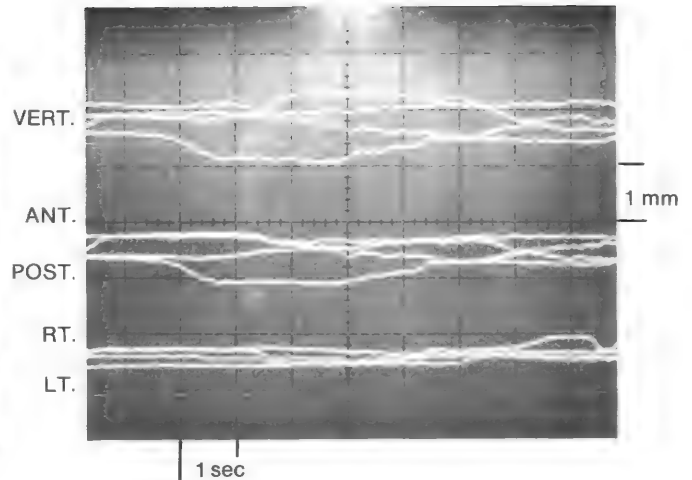
The mandibular motion at the right of the graph is of the subject tapping his teeth together three times. Note in this action there is minimal lateral slide into or out of centric occlusion. Often the first contact of the teeth stimulates the periodontal proprioceptors to bring more accuracy to succeeding closures.

Another reason to repeatedly tap the teeth together is to ascertain that the patient goes completely into intercuspation with the posterior teeth. Sometimes the first closure is on the anterior teeth only. This is indicated on the graph by the repeated tapping coming to a varying depth of intercuspation.

Temporomandibular Joint



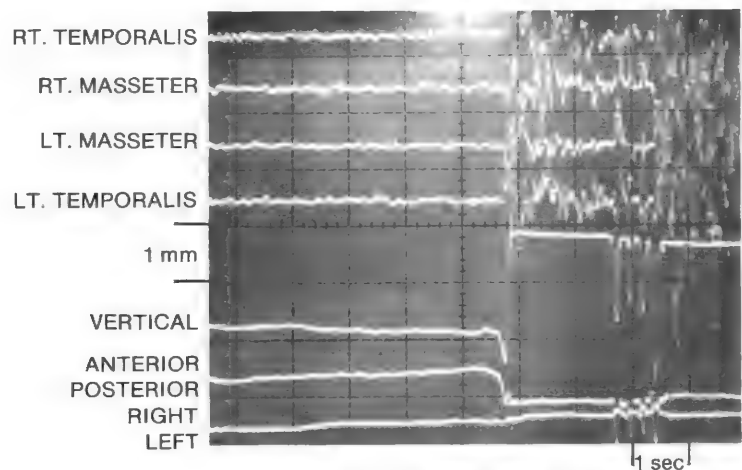
11—28. Stable rest position recorded for 40 seconds.



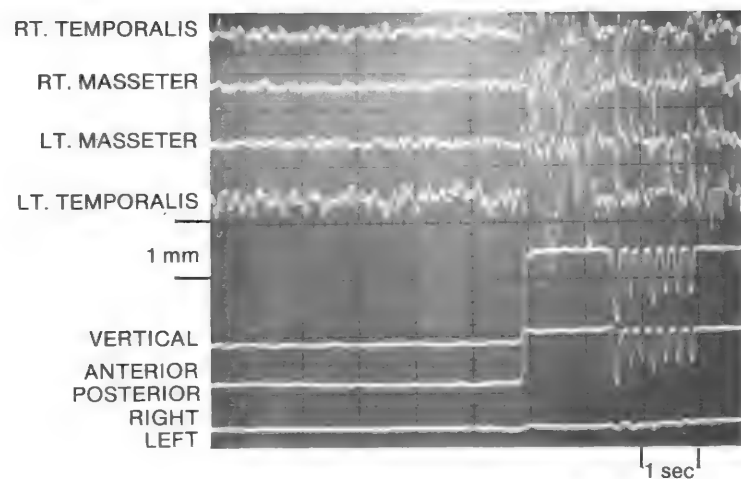
11—29. Unstable rest position tracing gives three dimensions of movement.

The storage feature of the oscilloscope is capable of monitoring the stability — or lack of it — on a continuous basis when the patient is in the rest position. In figure 11—28 there are four traces overlapping each other. Note in 40 seconds that the position of the mandible does not vary in excess of .2 mm. In figure 11—29 the musculature is tense and the rest position unstable. In the vertical dimension there are 1.3 mm of deviation in 40 seconds, .9 mm deviation in the anterior-posterior dimension, and .5 mm deviation in the lateral activity.

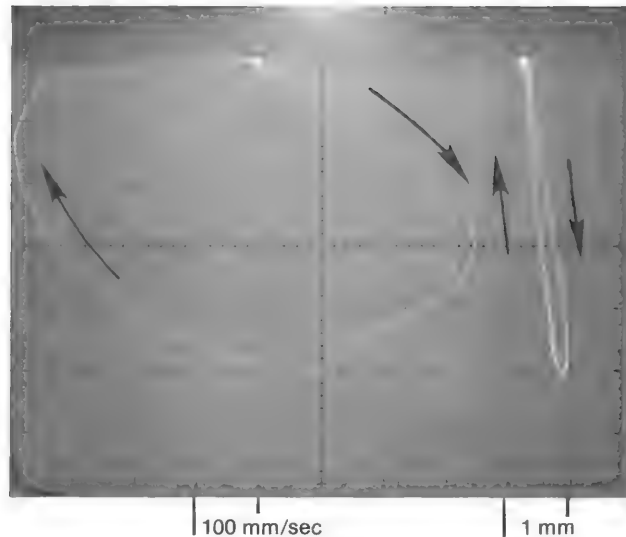
Four leads of electromyography can be recorded simultaneously with the mandibular position. Graph 11—30 illustrates minimal EMG activity of the anterior temporalis and masseter muscles with a relatively stable rest position. In graph 11—31 the electrical activity is increased in the anterior divisions of the temporalis, even though there is a stable rest position. This indicates muscle hyperactivity when the muscles should be inactive. This is probably not the true rest position of this individual.



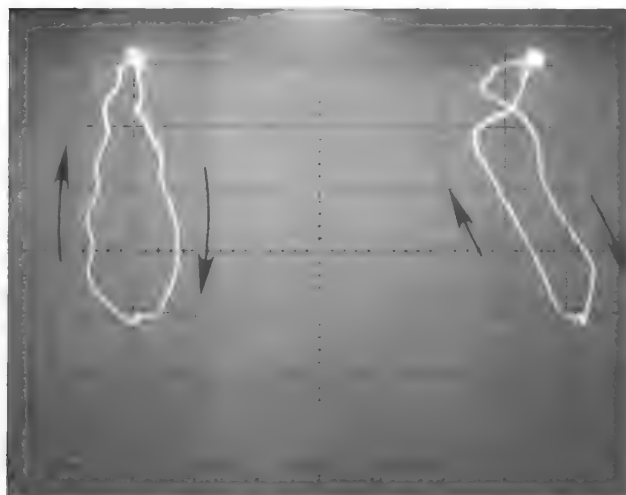
11—30.



11—31.



11—32.



11—33.

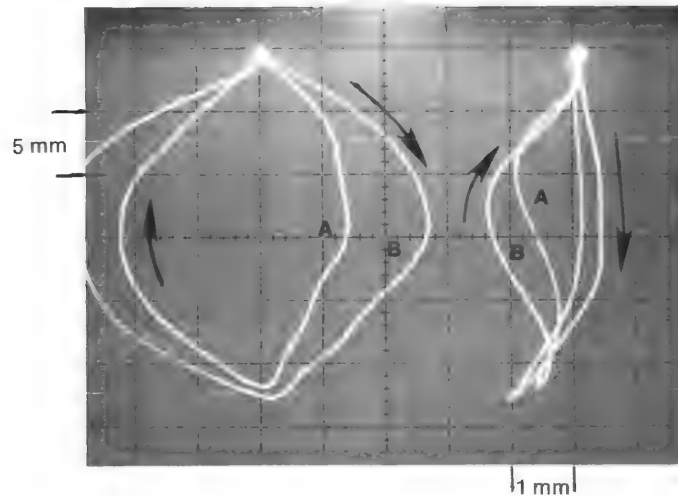
Vertical velocity of mandibular movement can be measured, which provides information to evaluate the smoothness of muscle synergism and the engram's influence on closing. In graph 11—32, the left tracing shows the extent of mandibular opening in the Y axis and speed in the X axis. As the mandible opens from centric occlusion, speed is gained to 340 mm/sec; it slows toward maximum opening to again increase speed to 400 mm/sec on closing. The speed then slows as centric occlusion is approached. Note the straight line close to the point of meeting centric occlusion. This reveals that the teeth met in intercuspation with a velocity of over 300 mm/sec. The smoothness of the curves of opening and closing indicates good neuromuscular coordination and TMJ function.

The right trace in graph 11—32 is the same as in graph 11—26, with the exception that lateral move-

ment has been magnified to 1 mm per division. This individual tested negative for TMJ dysfunction by AK methods.

Graph 11—33 depicts a subject with crepitation and disturbance in the neurologic pattern and muscular function, as revealed by applied kinesiology examination. Note the slowness of movement and lack of smooth curve on opening. Maximum velocity on opening was about 70 mm/sec; during closing it was 90 mm/sec. Normal velocity is 300 to 400 mm/sec. The frontal tracing on the right shows a left deviation of the mandible on opening to cross to the right and cross back to the left on closing to finally move to the right into intercuspation. Both of these tracings reveal considerable neuromuscular dysfunction, which was also diagnosed with applied kinesiology methods.

Temporomandibular Joint



11—34. Trace A is open-and-close at a moderate speed. Trace B is open-and-close as wide and fast as possible. Note how the engram slows movement toward intercuspation. There is left mandible deviation of 1.5 mm on closing.

11—34.

Often when there is a prematurity present, the teeth will not come into centric occlusion with great force. This appears to be controlled by the engram controlling closure. Graph 11—34 has two traces; one is of opening and closing at a moderate velocity, and the other is as wide and fast as possible. Note how the velocity is reduced considerably toward intercuspation. The engram appears to protect the teeth by not allowing a rapid closure, which would cause additional trauma to the teeth having premature contact. Encouraging the subject to speed up the closing, which overrides the engram control, typically causes pain in the teeth or in the temporomandibular joint. The trace on the right shows .4 mm of deviation to the right on opening, and 1.5 mm of deviation to the left on closing.

There are many additional uses of the Mandibular Kinesiograph for research and clinical evaluation. Some of these will be described later as the instrument is used to evaluate applied kinesiology techniques. The instrument appears to be the optimum approach for measuring mandibular function with current technology. It is accurate from 0 mm at intercuspation to a maximum of 0.5 mm at worst error. The area of greatest concern for measurement accuracy is at intercuspation, where the instrument has its greatest level of accuracy. The quantitative accuracy diminishes as the mandible moves further away from intercuspation. Computer processing is available for the instrument which reduces the worst error anywhere within the clinical space to 0.1 mm. Even without computer processing,

the instrument is qualitatively and quantitatively useful for research and diagnosis, without the need for further correction.⁵⁷

Jankelson uses the instrument in combination with his Myo-monitor⁸⁴ to obtain occlusal registration, rest position, and for other dental techniques.^{41, 56} Our AK use of the instrument varies from that of Jankelson.

Attachment of the magnet to the lower central incisors creates some problem in the instrument's use in applied kinesiology. In some individuals tested, the attachment of the magnet disturbs some or all of the muscles evaluated by manual muscle testing. This is apparently due to the magnet's effect on the meridian system, which seems to be electromagnetic in nature. To determine if the magnet changes the mandibular movement to give erroneous information, a study was done in our laboratory using a modified Dombrady¹⁹ method. Small electric lights were attached to the forehead and to the mandible. The lightbulbs and their associated wires weighed less than .62 gm, and the wires were suspended to avoid hanging from the mandible, causing an increased weight to be attached to it. The lightbulbs were turned on as the camera shutter opened and mandibular movement began. Movement of the light on the mandible traced a photographic record on the film throughout mandibular motion. Stability of the light mounted on the forehead insured that the subject was not moving his head with mandibular motion. This record of mandibular movement was photographically enlarged to allow an accuracy of

measurement to 0.1 mm and then compared with tracings done on the Mandibular Kinesiograph, with special critical analysis given to movement into and out of centric occlusion. The patterns of the two methods of measurement coincided, indicating that the magnet used in the MKG analysis did not adversely affect the measurement of the mandibular movement by that method.

When using the Mandibular Kinesiograph with applied kinesiology methods, it is necessary to record mandibular activity and then remove the magnet and sensor array to evaluate and treat the patient with applied kinesiology techniques. After treatment the magnet and sensor array are replaced, and the patient is re-evaluated on the Mandibular Kinesiograph for changes which may have taken place as a result of the applied kinesiology treatment. The sensor array of the MKG can be repeatedly calibrated to the patient with accuracy by experienced operators.⁵⁷

Use of the Mandibular Kinesiograph has helped put into perspective the changes which take place in the stomatognathic system as a result of applied kinesiology examination and treatment. It has also helped to understand the effects of equilibrating the occlusion to the different positions of centric relation, and to determine from a chiropractic viewpoint when dental consultation and treatment are necessary to effect permanent corrections in the stomatognathic system. An additional benefit the MKG has accomplished in applied kinesiology is documenting the changes which take place in mandibular movement from remote factors, such as foot and pelvic subluxations. At this writing the Mandibular Kinesiograph has been used in evaluating applied kinesiology treatment for a relatively short time. It has already improved examination and treatment techniques, and is expected to considerably enhance future long-term research projects.

Class of Lever

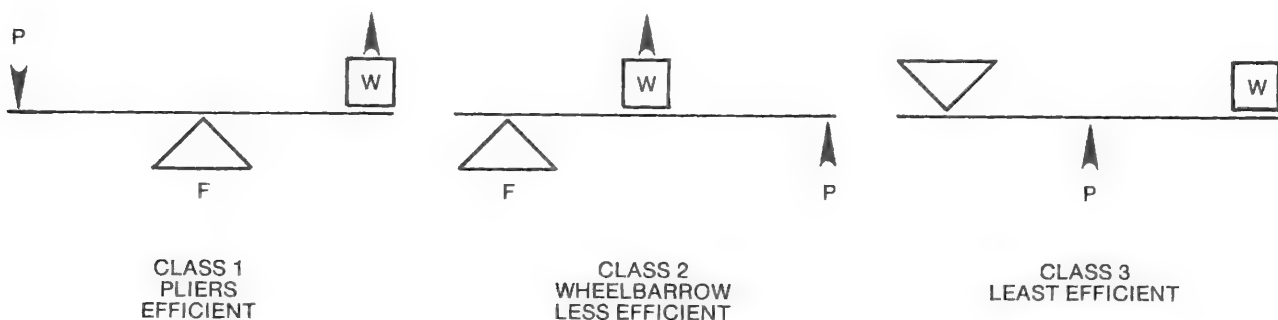
The lever classification of the masticatory apparatus varies according to the action taking place. Some authorities classify the temporomandibular joint as a lever of the third order. This is applicable in the stage of mandibular action which is limited to hinge motion without translation, but the function of the total mechanism is much more complicated than that.

A lever obtains its classification by the relationship of the fulcrum to the weight and power. The Class I lever is the most efficient, while the Class III is the least efficient.

The abilities of tissues of the dentition and the TMJ to withstand force make it necessary to understand how forces are applied to these structures. When the mandible closes freely, there is little force produced by the mandibular closers. It is only

when there is occlusal contact, or a rigid structure is between the teeth, that great power is produced by the muscles. This is when heavy stresses are placed into the structure.

The masticatory apparatus functioning as a lever of the third order requires two considerations. First is whether tissues are designed for this type of stress, and second is the efficiency of the mechanism if it is a lever of this type. In addition to evaluating TMJ and mandibular motion, it is necessary to consider stresses applied to the teeth during mastication. Some consider that the masticatory apparatus should be maintained as a lever of the third order, both unilaterally and cross-arch, in order to reduce stress on the teeth.⁴⁹ The normally functioning tooth is anchored solidly in the alveolar tissue by the

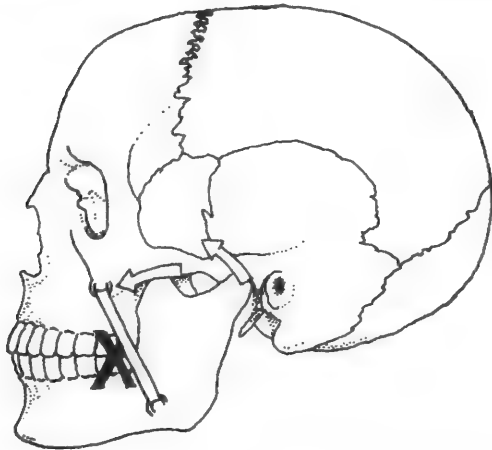


11—35. Classification of levers.

Temporomandibular Joint

periodontal ligament, which provides a shock absorber mechanism. The teeth are also protected by the opening reflex provided by the periodontal ligament proprioceptors and nociceptors.^{46, 82, 89} This seems to indicate that the teeth are structurally designed for and protected from the normal stresses present in mastication. This does not mean that they are structurally capable of the stresses present in malocclusion.

The other stress-bearing structures — the temporomandibular joints — have already been shown to be poorly adapted for direct weight bearing; they are



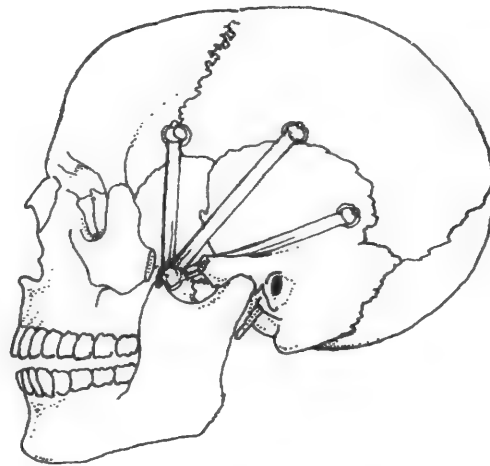
11—36. Forces developed without temporalis action (after Robinson¹⁰³).

more applicable to stresses of a shearing nature. As will be seen, this is the type of stress present when the teeth are the major weight-bearing structure as present in normal occlusion. The fossa is particularly susceptible to damage from weight bearing when the condyle is in the most retruded superior position. In an AK analysis this suggests an improper stabilization of the mandible by the temporalis, with hypertonic posterior fibers present either uni- or bilaterally.

The second factor of consideration is the efficiency with which the joint functions as a lever of the third order. The distance from the fulcrum of the TMJ to the load of a food bolus between the teeth causes a considerable waste of energy, making this proposed mechanical system generally very inefficient.⁴³

Wilson¹⁴⁰ and Robinson¹⁰³ proposed that the temporomandibular joint does not act as a lever of the third order during mastication and the occlusion of swallowing. This is based on alignment of the fibers of the masseter and the internal pterygoid muscles which pull on the mandible in a superior and an

anterior direction, causing intercuspatation which tends to bring the condyle toward the articular eminence.¹⁶ The posterior direction of the temporalis muscle, when taken in total context, balances the forces of the masseter and internal pterygoid muscles so that the massive force exerted in strong intercuspatation is taken by the dentition and not the TMJ. Electromyography shows the time sequence of muscle contraction during the chewing of soft or hard foods; the masseter muscle contracts first, followed by a stabilizing action of the temporalis muscle. The delay of temporalis activity is shorter when chewing hard foods as opposed to soft.¹²¹



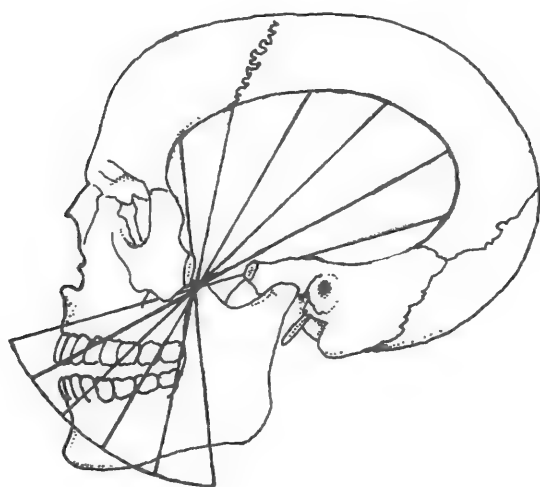
11—37. Synergistic action of temporalis combined with action in 11—36 gives a net result of a non-lever action (after Robinson¹⁰³).

Wilson and Robinson's theory is supported by Moyers'⁸⁰ electromyographic study in which he states, "The direction of the muscle pull, the sequence of the contractions, and the action of synergists all show that the resultant force is in the denture. There can be no lever action when the resistance is directly in line with the resultant of the applied power; such is the case in the mandible. The teeth and their supporting structures thus bear the heavy stresses for which they are designed and the joint is normally free of heavy strain." This analysis supports the theory that the temporomandibular joint is not designed to be a strong weight-bearing joint, as indicated earlier in this chapter.

In an anthropologic study, Gingerich⁴³ points out that in most reptiles, birds, and mammals the lower jaw is generally free to translate fore and aft with respect to the cranium. This takes place by either of two mechanisms. Translation may be from a sliding action of the condyle — such as in man — from the temporal fossa onto the articular eminence, or by a

two-jointed jaw suspension as is present in most lizards and snakes.

In man some fibers of the temporalis muscle are always in alignment with a food bolus placed between any of the teeth. Since the mandible is only in contact with the skull at the point of the food bolus and the condyle, components of reaction force can only occur at these two points. The well-lubricated condyle is free to slide posteriorly with contraction of

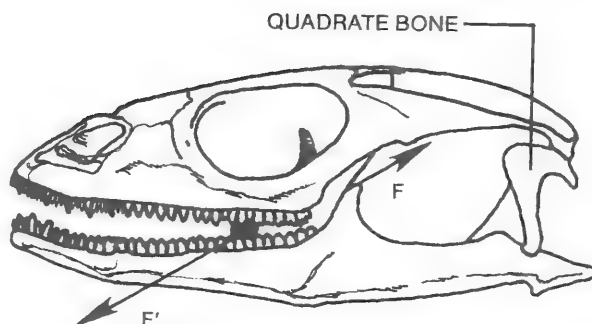


11—38. There are temporalis fibers in alignment with all of the teeth, providing a crushing mechanism (after Gingerich⁴³).



11—39. The fibers of the temporalis provide for stabilization and control of the condyle in the mandibular fossa.

Temporomandibular Joint



11—40. Quadrate bone allows free translation anteriorly and posteriorly. The muscle force (F) is equal to the bite force (F'). After Gingerich.⁴³

the temporalis muscle. Minimal force would thus be applied at this area, with the maximum amount of pressure applied to the food bolus with efficient use of the muscle. McCall and Swinyard⁶⁹ emphasize this by stating, "As a matter of fact, anatomical varieties and functional considerations indicate that the mandible is not a lever at all but a crushing mechanism."

The two-jointed jaw suspension of lizards and snakes uses an additional bone to obtain the sliding action. This bone — the quadrate — is located between the mandible and the skull for rotation, to allow free translation anteriorly and posteriorly.

The ability of the TMJ to translate and the variable synergistic action of the muscles of mastication provide considerable versatility in mandibular action. There is a different workload during incising and trituration as compared with biting hard with the posterior molars. The type of motion and class of lever action of the temporomandibular joint varies with different circumstances and how the muscles act in variable synergism. During function the relationship of the condyle with the fossa varies from posterior to anterior and lateral. Because of this flexibility, any attempt to classify mandibular activity as one lever type is oversimplification of the activity.¹³⁵ The importance of muscular action in mandibular motion is emphasized by the ability of the masticatory system to function in the absence of condyles.⁴⁸ Kawamura⁶¹ reports, "Even the patient who has lost the temporomandibular joint by surgery or congenital anomaly may move the mandible fairly well by means of jaw muscle action. However, the range of motion will be somewhat limited; mandibular movements will not be able to adapt well to the emergency situation and the delicacy of mandibular movements will be lost."

TMJ Internal Derangement

The temporomandibular joint, like any other joint of the body, is susceptible to internal derangement which may take any of many forms. The joint may be involved with arthritis, inflammatory processes, remodeling, disc degeneration, or disc dysfunction.⁸

Posterior Capsulitis

Posterior capsulitis refers to inflammation or pain behind the condyle.²⁵ The condition is always associated with imbalance of the masticatory muscles as observed in applied kinesiology. The temporomandibular joint area will therapy localize¹²⁹ with no movement of the joint or with the jaws in any position, including maximum intercuspation.

When in the rest position, the mandible will deviate away from the affected side. There is a positive response when the examiner moves the mandible so that pressure on the posterior aspect of the temporomandibular joint increases. The examiner grasps the mandible and has the patient move it laterally to the affected side. This moves the condyle posteriorly in the fossa, and the examiner then presses gently to increase the pressure in the fossa. A positive response is increased pain; if there is no response, a more forceful pressure may be applied. Very often the external pterygoid is found to be hypertonic on the side of involvement. The posterior fibers of the temporalis muscle are also often hypertonic in this condition. The applied kinesiology approach is to evaluate for and treat temporomandibular joint dysfunction as described in Chapter 14. Farrar²⁵ describes eliminating "interceptive contacts" as a therapeutic approach. He states, "Radical or extensive occlusal grinding is not necessary to relieve the pain of posterior capsulitis."

When there is positive evidence of inflammation in the TMJ, such as in posterior capsulitis, laboratory procedures are indicated. They should include a CBC and arthritis differentiating tests, such as RA latex fixation, uric acid, sedimentation rate, and roentgenologic study.

Arthritis

Any of the various types of arthritis can affect the temporomandibular joint; the most commonly involved is degenerative joint disease (osteoarthritis). As in other joints of the body, the condition is brought on by chronic stress in the joint, particularly when its tissues are not of optimum quality. Ricketts⁹⁹ studied 463 individuals in a Veterans Administration hospital population for degenerative joint disease of the TMJ. There was advanced breakdown and definite concern in about one in eight males, and approaching one in three females. The males' average age range was in the fifth or sixth decade of life, with the older group being edentulous. The females' average age was forty-five years. The higher incidence of degeneration in the female is probably due to hormonal differences.

There are two primary concerns for treatment of degenerative joint disease of the TMJ. First, stress in the joint must be relieved.⁹⁵ Although this does not cure the existing condition, there is evidence that reparative remodeling takes place when stress is removed.^{7, 8, 74} Constant remodeling of the joint takes place as an adaptive process. Remodeling becomes a pathologic process when the stress is so great it is no longer a functional adaptation. Stress in the joint is usually caused by muscular imbalance, loss of vertical dimension, and/or disc dysfunction.

The second factor responsible for degenerative joint disease of the TMJ is poor health of the soft tissues and bone of the joint. This can occur in combination with stress to the joint, or individually. The usual physiologic disturbance causing poor quality of the joint tissues is in protein and calcium metabolism. (The nutritional and other approaches used in applied kinesiology in the treatment of arthritis are discussed in Volume V.)

Rheumatoid and gouty arthritis are systemic conditions. There is not a high incidence of their involvement with the temporomandibular joint. It is obvious they must be differentially diagnosed, along with other more uncommon conditions, such as osteochondritis and other conditions listed in Chapter 14.

DISC DYSFUNCTION

Normal function of the temporomandibular joint depends upon muscular balance and the type of occlusion present. Normal TMJ function, of course, includes normal disc function. One wonders why

there is so much consideration given to terminal hinge centric relation coinciding with centric occlusion, but so little consideration given disc function when it is so important to TMJ activity. A close

evaluation of disc function shows the need for centric occlusion to be in harmony with physiologic centric relation. This is anterior to the terminal hinge centric relation. Equilibrating the occlusion so that centric occlusion coincides with centric relation in the most retruded superior position brings the condyle posterior; this predisposes an individual to the development of disc problems.²⁸

Along with proper occlusion, muscle balance is primary for the disc-condyle complex to function normally. Joint function anywhere in the body depends upon balanced muscle function. Although ligaments limit the range of a joint's motion, in the final analysis it is the muscle system which holds the skeletal system together. In the presence of muscle imbalance, the soft tissues and bones of the joint receive the stress. This relates to the well-known phrase, "When muscles and bone war, bone never wins." Kawamura⁶¹ points out the importance of the external pterygoid muscle heads acting harmoniously to move the articular disc in translation, supporting it against the articular eminence. McNamara⁷² further points out that the inferior head acts in opening movements, and the superior head stabilizes the disc against the articular eminence during closing movements of the mandible.

The anatomy of the TMJ and disc was discussed earlier in this chapter. The disc's posterior attachment to the capsule is important in maintaining its orientation in the TMJ complex. Most disc dysfunction is a result of the disc losing solid posterior attachment and disrelating with the condyle. Disc movement is anterior or anterior and medial; rarely, if ever, does it displace posteriorly.³⁵ Displacement of the disc relates with clicking, popping, and locking or limitation of temporomandibular joint movement. Ireland⁵⁰ was the first to make this observation. He noted that generally when the TMJ clicks and pops, it is not locked; when it is locked, the TMJ does not click. This observation led to the conclusion that these conditions are caused by anterior displacement of the disc with the condyle.

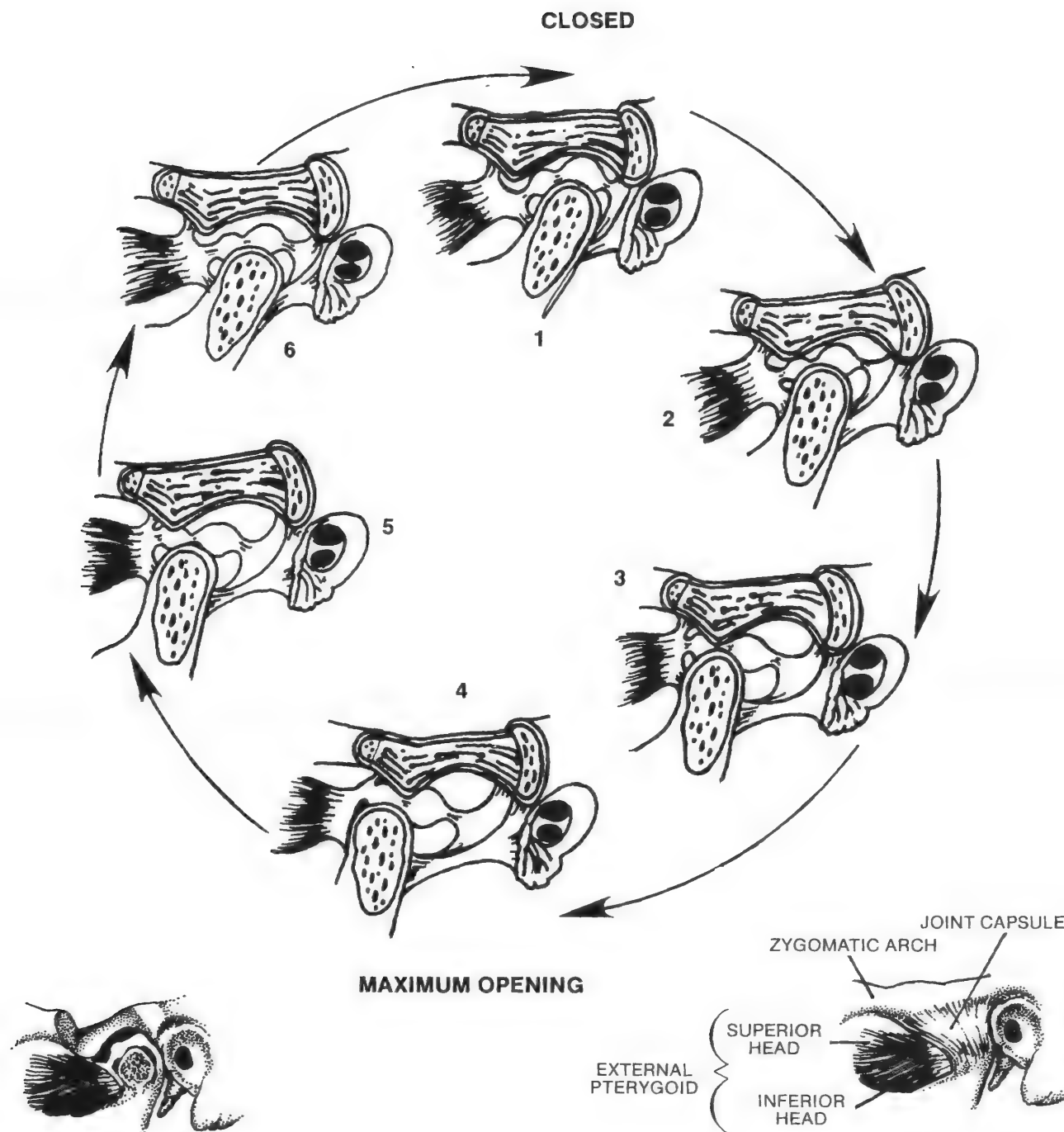
Farrar points out in many writings^{28, 29, 30, 31, 35} that precise relationship between a retruded superior centric relation and centric occlusion is unnatural; it positions the condyle posteriorly to set up a possible iatrogenic anterior disc condition. He defines an optimum centric relation as "... the most superior position of the condyles at which a hinge axis movement can be recorded, *provided the discs are not displaced.*" Ricketts,¹⁰⁰ discussing type B distal displacement of the condyle, states, "Perhaps this type is the most misunderstood and ironically is the very kind that has been produced by the dentist; in many cases it is iatrogenic. Therefore the astute

clinician should be aware of this condition or at least recognize it when he has produced it. He should listen to and be guided by what the patient is trying to tell him in certain of these instances."

Another possible cause of a posterior position of the condyle is hypertonicity of the posterior fibers of the temporalis muscle. Moyers⁷⁹ observed on electromyography that in individuals with normal dentofacial development, there is an even state of "tonus" in all divisions of the temporalis muscle, even though the muscle has triple innervation. In many cases of mandibular retrogression there are greater contractions from the posterior fibers of the temporalis muscle than from the anterior fibers. Jarabak⁵⁹ has demonstrated electromyographically that there is spontaneous hyperactivity of the posterior fibers of the temporalis muscle with disc dysfunction. Hyperactivity was also occasionally recorded from middle fibers, but not from the anterior fibers. Other studies also show the posterior fibers of the temporalis muscle active in retraction of the mandible.^{5, 65, 83}

Earlier evaluation of disc position was done by flat x-rays taken in the open, closed, and rest positions. There are two criteria for determining disc displacement. One is limitation of forward condylar movement to 30%-50% of normal when in the open position. Second, the condyle is inferiorly displaced in the open position as observed by an increase in the interarticular space between the condyle and the anterior slope of the fossa.^{26, 29} With rare exceptions there is disc involvement when there are bilateral asymmetric TMJ spaces observed on x-ray.¹³¹ Further description of x-ray analysis of the temporomandibular joint for disc displacement is presented by Weinberg^{132, 133, 134}

A more recent method for evaluating disc position is by arthrography.^{9, 33, 137, 138} The procedure requires inserting a contrast medium into the joint space(s) to outline the articular disc and its attachments; this enables identification of the precise position and morphologic structure of the soft tissues not ordinarily seen on x-ray. This procedure is indicated for persons being considered for disc surgery, which is done after the more conservative approaches, described later, have been attempted and failed, and the condition remains or progressively worsens. Techniques of the procedure vary; some inject the radio-opaque substance into the superior and inferior spaces, others inject only into the inferior space. Serial x-rays are then taken with the jaws closed and at various degrees of opening. This allows a visualization of the disc's relation with the condyle through the range of motion. The action of the disc in opening click, reciprocal click, and opening lock, described next, is determined from studies of the disc with arthrography.

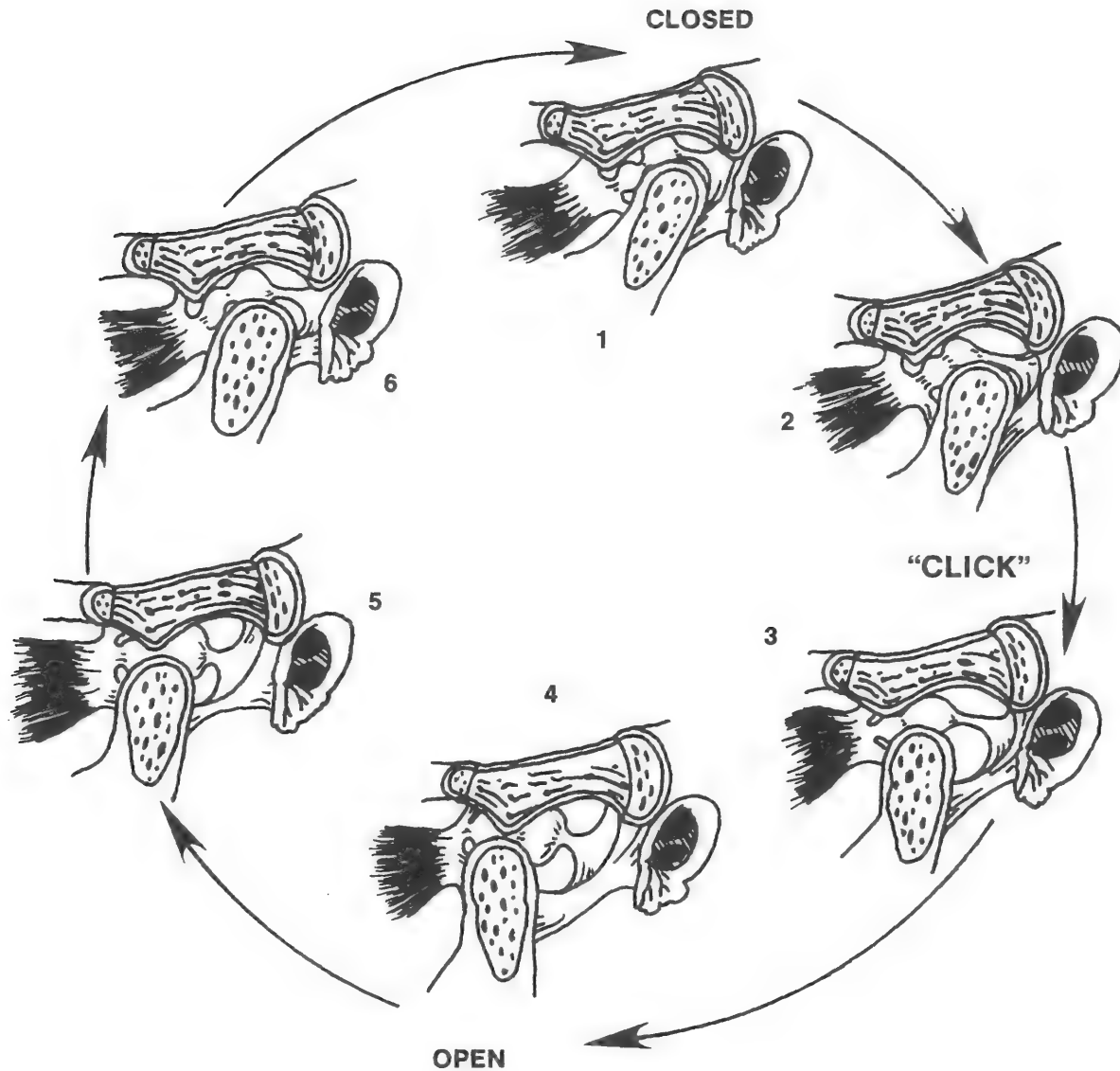


11-41.

Normal Disc Movement

Figure 11-41 illustrates a disc in its normal action with the condyle through maximum opening and closing. Number 1 is closed in the rest position. Upon opening, the inferior head of the external pterygoid moves the condyle and disc forward to

begin translation. This action continues to position 4, which is maximum opening. Note that the disc remains with the condyle through the entire action. During closing, the superior head of the external pterygoid appears to stabilize the condyle and disc mechanism as it returns to the articular fossa.



11—42.

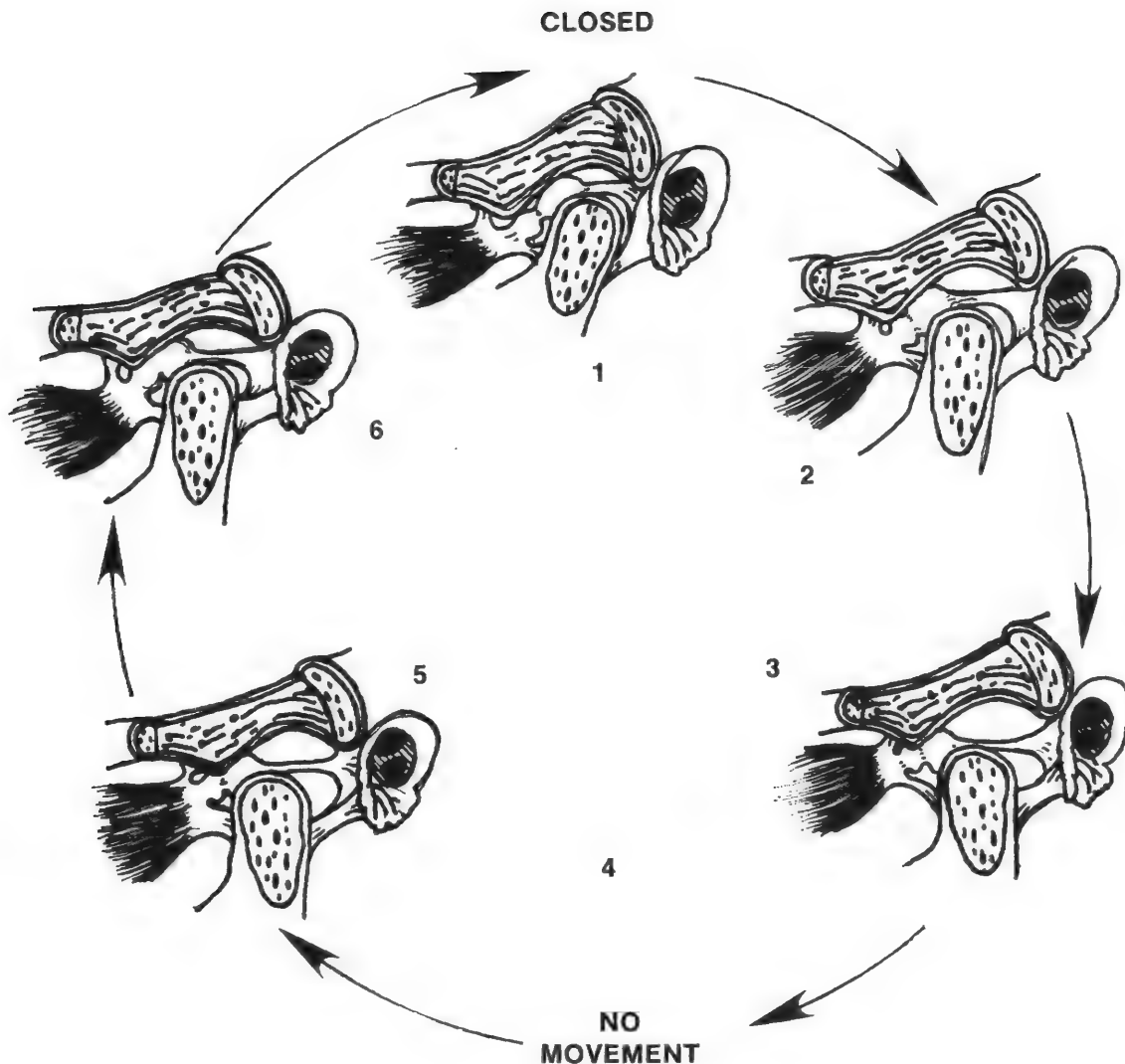
Opening Click

In the opening click the condyle is in a posterior superior position, and the disc is displaced anteriorly. As opening begins at position 2, the disc is pushed forward by the condyle as translation begins. As movement continues to position 3, the posterior attachment of the disc tightens; instantaneously the disc snaps into its normal position. The snapping of the disc is the pop or click heard in jaw motion. The disc remains with the condyle through the balance of translation to maximum opening. In closing from positions 4 to 6, the disc rides with the condyle to the

closed position. The condyle slides off the disc in an unobstrusive manner to return to its retruded superior position.

Reciprocal Click

The reciprocal click is similar to the opening click, with the exception that in closing the condyle snaps off the disc to create a closing click. There usually cannot be a closing click without an opening one, since the condyle and disc must snap into a proper relationship before the condyle can snap off the disc.



11—43.

Opening Lock

The opening lock is a more extensive condition than the opening click. In position 2, the disc is pushed forward of the anteriorly moving condyle and is bunched up so that it cannot snap into proper relation with the condyle. The posterior attachment of the disc tightens and movement stops, producing the opening lock. Ricketts¹⁰¹ calls this a permanently anteriorly displaced disc (PADD). With chronicity, the posterior attachment stretches or is traumatized so that the disc may be pushed further forward, allowing a greater amount of jaw opening. The fact that the patient with an opening lock obtains a greater range of motion in opening does not usually indicate the condition is improving; rather, the posterior attachment is becoming less stable.

Disc dysfunction usually goes through several stages. The first symptoms are often crepitation and edema in the joint, caused by joint stress which creates poor organization between the disc, the capsule, and the articular fossa. Joint stress can result from anything which upsets the homeostasis of the stomatognathic system. A muscular imbalance anywhere in the closed kinematic chain can change occlusion. Malocclusion can also develop as a result of cranial faults or from improper equilibration. Of course, as we have seen, any one of these factors starts a vicious circle. Malocclusion can increase the cranial faults, which increase the muscular imbalance and continues to change the occlusion, and on and on the series of events go, in any direction. As the disc becomes more disoriented with the condyle,

the crepitation changes to specific clicking or popping. At first the opening click is in the early portion of translation.³³ As the posterior attachment of the condyle becomes more unstable, the click develops later in the opening movement. This progression may take from months to years. Eventually the disc remains in front of the condyle, and an opening lock develops. Again, as the posterior attachment becomes more unstable, the opening lock progresses to allow greater opening by further movement of the condyle in translation. Although the above sequence is usual, it is not always in this order. It may go from opening click to reciprocal clicking to opening lock. A person may begin with reciprocal clicking to eventually move into opening click and then opening lock, or the condition may begin with

opening lock.

The type of disc disrelation with the condyle is reflected in the terminology which relates to the disc. A disc displacement occurs when the disc is anterior to the condyle but can be reunited with the condyle, which must move inferiorly to slip beneath the posterior band of the disc before moving forward. Disc dislocation takes place when the disc is anterior to the condyle, preventing its anterior movement and limiting translation.²⁸

Prognosis for conservative treatment improves if the clicking or locking occurs during the early stages of translation. Clicking or popping later in the opening cycle indicates more severe loss of integrity of the posterior attachment.

CONSERVATIVE TREATMENT

There are several conservative approaches to treating the clicking, popping, cracking, or locked TMJ. Two steps must first be accomplished. These are to (1) balance all muscles in the stomatognathic system, especially leveling the head, and (2) evaluate and correct occlusion, if necessary. These steps should be taken in the order given since muscular balance has a major influence on the occlusion.

Usually the first step in balancing the muscles of the stomatognathic system is to evaluate and treat, if necessary, the major postural muscles of the closed kinematic chain — the sternocleidomastoid, upper trapezius, and deep flexor and extensor muscles of the cervical spine and head. They are often involved in TMJ problems¹⁰⁵ and should be evaluated in various postural positions since many times imbalance relates with weight bearing. If disturbance in the muscles is present with weight bearing, the feet, pelvis, spine, and other major weight-bearing structures should be evaluated and treated, if necessary. Quite often the sternocleidomastoid and upper trapezius imbalance is caused by cranial-sacral primary respiratory dysfunction, because these muscles receive some of their nerve supply from cranial nerve XI. In returning balance to these muscles, there is often a major change in many of the muscles of the stomatognathic system. This is true whether the muscles are improved from cranial fault correction or from some other treatment approach.

Treatment of the cranium may directly influence the muscles of mastication, muscles of the orofacial complex, and muscles of the hyoid by improving

function of cranial nerves V, VII, IX, and XII. Other forms of treatment to the major postural muscles of the closed kinematic chain may accomplish the same results as direct cranial correction. This is because they pull on the cranium. When balanced, they may correct cranial faults and thus reduce cranial nerve entrapment. Cranial correction may also cause major changes in the occlusion which can be positive or negative. The next step in muscular balancing is to evaluate the muscles of mastication as described in Chapter 14.

Prior to evaluating the masticatory muscles, the periodontal proprioceptors should be examined. If they are being improperly stimulated from an aberrant neurologic tooth condition, the muscular balance may be adversely influenced (see page 265).

Particular attention should be given to the posterior fibers of the temporalis muscle. It is often noted with applied kinesiology techniques that these fibers are hypertonic. Farrar and McCarty³³ state that "... the posterior fibers of the temporal muscle are more susceptible to stretch injury than any other muscular components of the stomatognathic system." They relate this to excessive forced opening of the mouth, particularly during general anesthesia. These posterior fibers tend to position the condyle posteriorly, setting up an environment for the disc to be displaced anteriorly.

The posterior fibers of the temporalis muscle can be evaluated by therapy localization. Positive therapy localization may be to a neuromuscular spindle cell or to the general area of the muscle when there is

Temporomandibular Joint

fascial dysfunction. A modified muscle stretch reaction is used to test the muscles of mastication. The patient therapy localizes over the muscle to be examined, and a previously strong indicator muscle is tested for weakening. If it weakens, it is probably not due to a muscle stretch reaction. Evaluate the muscle for neuromuscular spindle cell involvement or some other factor that is causing the positive therapy localization. If there is no weakening to the therapy localization, the mandible is passively opened to the limit of motion, imparting a moderate stretch to the muscle fibers. A previously strong indicator muscle will weaken in the presence of a positive response. The treatment indicated is spray and stretch or fascial release technique.

The patient can be tested for normal operation of the temporal tap to further evaluate for hypertonicity of these fibers. The temporal tap will not operate in its normal manner with the hypertonicity, but will return to normal after release of the muscle. (See Volume I for a more complete description of muscle proprioceptor, muscle stretch reaction, spray and stretch, fascial release, and temporal tap techniques.)

The external pterygoid appears to be important in helping maintain the disc-condyle relationship during movement. Evaluation of the external pterygoid and its treatment are discussed in Chapter 14. There may also need to be evaluation and treatment of the hyoid muscles, described in Chapter 13.

The final consideration in muscle balance is the orofacial group of muscles. Most often they will be functioning normally, following the previous corrections. If imbalance remains, its cause is localized in the muscle proprioceptor, fascia, or trigger point, or requires myofunctional therapy to change habit patterns or build atrophied muscle, discussed in Chapter 15.

This short description of balancing the muscles of the stomatognathic system may seem extensive and excessive, requiring a considerable amount of the physician's time. In reality, the examination and required treatment are quickly accomplished by the skilled applied kinesiologist and make a dramatic change in the function of the entire complex. The neophyte in applied kinesiology should master one step at a time. It will quickly become obvious how all areas interrelate; one division cannot be adequately corrected without giving attention to the total complex.

Occlusion is evaluated next. If there is malocclusion, especially one that forces the condyle into the most retruded position, successful conservative treatment to the TMJ disc is severely compromised.

The condyle should be located in physiologic centric relation, coincident with centric occlusion. As

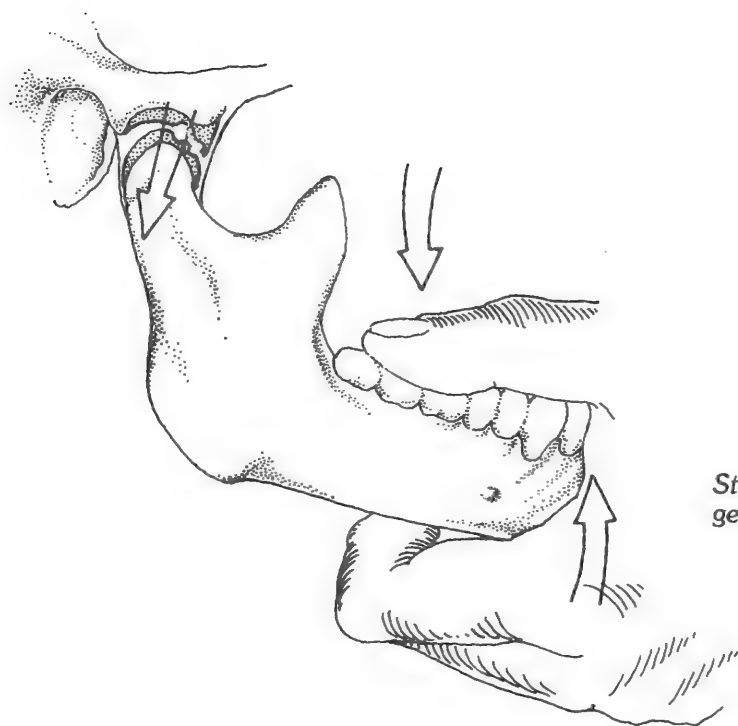
previously noted, the physiologic centric relation position can only be determined in the presence of balanced, relaxed muscles. Applied kinesiology concepts of occlusal harmony with the rest of the stomatognathic system are discussed in the next chapter.

The disc manipulation to be considered now is usually not successful if the previous balancing of the stomatognathic system has not been accomplished. The objective of conservative disc treatment is twofold. First, a normal position of the condyle must be obtained and, second, harmony of the disc with condylar movement must be regained.

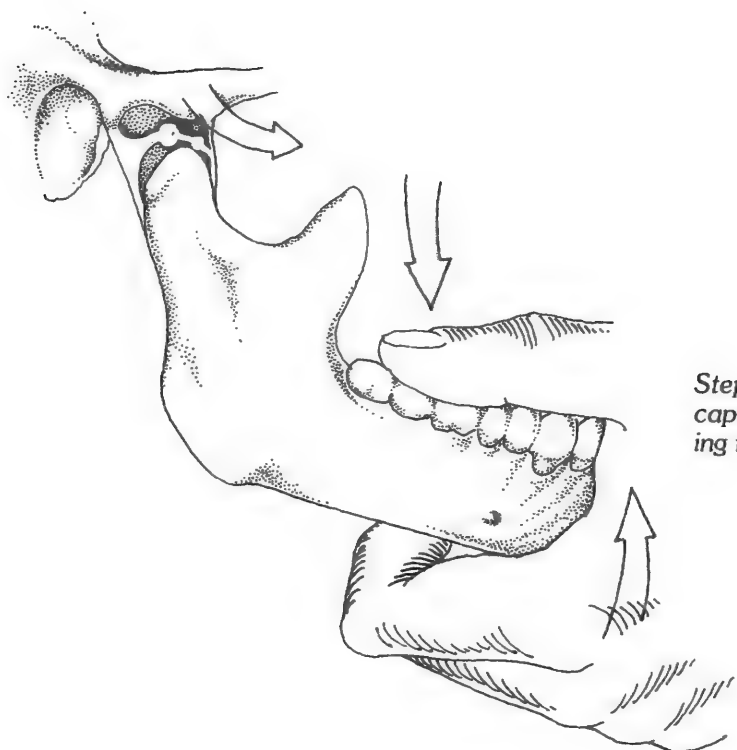
The effort is to manipulate the condyle and disc in such a manner that they regain their juxtaposition. The improved condylar-disc position will often unlock an opening lock.³² If both sides need manipulation, only one side is treated at a time.

The patient is usually supine, but he may sit with his head against a solid headrest. The procedure begins by the physician making thumb contact with the molars on the side of involvement. The lower anterior aspect of the chin is cradled by the remaining fingers. It is optional whether the physician uses both hands to contact the total mandibular arch. Gentle inferior traction is applied to the molars to inferiorly displace the condyle. This is enhanced by the physician applying superior pressure to the symphysis menti area with his fingers. This applies a cantilever-type action to the mandible. The inferior traction is continued until full relaxation and separation of the condyle are felt. The force is then directed anteriorly to the molar teeth, while inferior traction is maintained. The manipulation is done in a smooth, continuous, and controlled manner with only minimal quickness of movement. This control is necessary, especially when there is great looseness of the articular capsule, to avoid a dislocation of the condyle anterior to the articular eminence. In the case of an open lock, a distinct movement of the disc will most often be felt as it pops into place. There will not necessarily be an audible click in all cases of disc replacement. As noted, if both sides are involved, one side should be manipulated at a time. It may be necessary to have an assistant stabilize the patient's head, as there is often a tendency for the head to move with the manipulation, thus negating the forces being applied.

Immediately after the manipulation, a dental cotton roll or roll of 2" x 2" sponges should be placed between the molar teeth on each side so the patient will not bring the teeth immediately into intercuspation. The dislocated disc has become distorted, and it is necessary for the condyle to slowly be placed into the central portion of the disc to re-form to the shape



Step 1. Open the articulation by gentle, persistent inferior traction.



Step 2. Bring condyle anterior to capture the disc while maintaining the inferior traction.

11—44. Maneuver to recapture disc with the condyle.

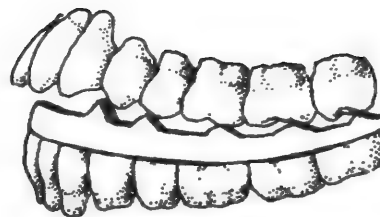
Temporomandibular Joint

of the condyle.³⁵ The patient is left in this position for five to ten minutes. When the prop is taken from between the molar teeth, the mandible is allowed to slowly move toward intercuspation. If the disc immediately returns to the abnormal position, there is probably malocclusion which must be dealt with dentally. This will be discussed in the next chapter.

After conservative treatment as described above, the patient is advised to not open the TMJ wider than the width of the thumb for two weeks. This helps allow the capsule and posterior attachment to more quickly regain integrity. The patient should be re-evaluated for muscular balance and the need for additional disc manipulation periodically during this time. If there is recurrent muscular imbalance, some factor contributing to the condition has not been found and corrected. If the muscular balance and cranial mechanism remain functioning normally but the disc needs continual re-manipulation, there is probably a malocclusion which must be treated. Probably if the conservative manipulative approach described to this point is unsuccessful, the disc will lose its normal position and the muscular imbalance and cranial faults will all return. If the physician treating to this point is not a dentist, then dental referral is necessary. Ideally the dentist will be familiar with the examination and treatment for balancing the muscles and correcting cranial faults already discussed. If not, it is necessary for the applied kinesiologist to work with the dentist in obtaining further correction. Clinical experience shows that treatment limited to disc manipulation and occlusion is not nearly as effective as correcting the muscular imbalances and cranial faults prior to the manipulation and occlusal correction.

Looseness of the capsular ligament is associated with disc displacement,³⁵ as is compromised integrity of the disc's posterior attachment to the capsule. Nutritional support in the form of manganese and products with raw bone concentrate helps improve the integrity of these ligamentous structures as observed clinically.

There are several dental procedures for re-positioning the mandible anteriorly. Farrar^{27, 35} describes a maxillary bite plane with steep inclines to re-position it. The bite plane is worn continuously, even while eating; the teeth are not allowed to come together at any time. When it is necessary to remove the bite plane, as when brushing the teeth, a mouth prop is inserted to prevent intercuspation during swallowing. The patient is re-examined to ascertain that the disc is maintaining its proper position. If not, it is re-manipulated; this may be required several times. Throughout this re-evaluation, the muscular balance and cranial mechanism are examined and



11—45. Exaggerated illustration of bite plane designed to reposition the mandibular dentition.

corrected, if necessary. The objective is to maintain the disc in its normal position in the presence of balanced musculature and cranial function. The bite plane is usually worn for six to eight weeks. When normal equilibrium of the disc, mandible, muscles, and cranium has been established, the bite plane is discarded. It will then be necessary to equilibrate the occlusion to this balanced physiologic centric relation. This may require only selective grinding, or it may require reconstruction.

In most cases, considerable cranial correction will be necessary. A mandibular bite plane is preferable in this case because a maxillary bite plane tends to lock the cranium. This preference is discussed more thoroughly in Chapter 12.

Another type of mandibular re-positioning device is constructed on a maxillary bite plane. A tapered projection is attached to the anterior palatal area of the bite plane to protrude inferiorly. As the mandible closes, the lower central incisors are forced anterior-



11—46. Mandibular repositioner. The mirror image gives an inferior view of the appliance.

ly, thus positioning the entire mandible anteriorly. The large projection interferes with tongue action, especially in speaking. As would be expected, it is tolerated by patients but not very well-liked. The inclined bite plane appears to function just as effectively, if not more so, and is a much more comfortable appliance.

If appliances are used to re-position the mandible anteriorly, care should be taken that it is not done

excessively. It has been observed that sometimes the mandible is moved so far forward that the centric relation position is anterior, toward, or on the posterior aspects of the articular eminence. This creates disharmony of structure that may be just as problematic as the retruded condyle. Anterior re-positioning is not a problem when the method used is muscular balancing and equilibrating the occlusion to that mandibular position.

SURGERY

Surgical intervention is usually indicated only after a thorough conservative approach has been attempted.^{17, 113, 116} One indication that some patients receive surgery when a conservative approach would suffice is that "... many cases show no evidence of gross or microscopic pathologic disorder ..." in the tissues excised during surgery.⁶² The surgery that appears most applicable is to re-position an anterior disc.⁷⁰ The most common internal derangement is an anterior disc requiring reconstruction of the posterior attachment.¹³⁶

A procedure described by McCarty and Farrar⁷⁰ to return the disc to its normal position includes removing a small amount of the condyle to provide room for the disc and avoid adversely modifying the relationship of the patient's occlusion. This surgery is correlated to the "... condition of the dental occlusion and in no case should surgery be per-

formed when the patient has an inadequate or unstable dental occlusal relationship with the posterior teeth on the same side."³⁵ Of patients seen with TMJ complaints, 71% were diagnosed as having an anterior displacement of the disc. Seventeen percent of all their TMJ patients eventually required surgery. This high percentage of correction by conservative treatment is without the benefit of applied kinesiology muscle balancing and manipulative techniques in their excellent regime. The conservative approach is described as "... repositioning prostheses, followed by occlusal equilibration, restorative dentistry, and/or orthodontics." The muscle relaxation techniques are described as medication, trigger point therapy, and exercises, including stretching procedures.³⁵ Condylectomy, meniscectomy, surgical recontouring of the condyle, and the insertion of prosthetic devices are rarely — if ever — indicated.^{18, 35}

Trismus

Inability to open the jaws is nearly always caused by an anterior dislocation of the disc. The procedures outlined above are first considered in differential diagnosis of trismus.

Although the disc is nearly always involved, it is observed in applied kinesiology that the primary factor may very well be a muscular imbalance which may seem quite remote from the condition at hand. Hypertonicity of the external pterygoid can cause the disc to be out of synchronization with the condyle. Other more remote disturbances in the closed kinematic chain can be primary. Immediate unlocking of the jaw can be the result of correcting a hyoid muscle imbalance, cranial correction, or even correcting the postural muscles within the stomatognathic system by comprehensive AK technique. As

with all other examinations of the stomatognathic system, remote possibilities outside the system must be considered.

Placing two dental cotton rolls between the upper and lower central incisors and allowing the patient to rest against this will sometimes remove enough muscular spasm to make it easier to proceed with the examination. Relaxation from the cotton rolls is usually accomplished within five to ten minutes.

Forcing the jaws open as is sometimes suggested is contraindicated in cases of trismus. Damage to the disc and other structures may result. As is always the case, working *with* the physiology rather than against it is the optimum procedure.

Dislocation

The appearance of a dislocated jaw is very characteristic and easily recognized. The patient is unable to close his mouth, the mandible is deviated to one side if the dislocation is unilateral (which is most often the case), and palpation of the TMJ reveals a depression posterior to the condylar head, just anterior to the external auditory meatus.

The dislocation is generally not the result of trauma; it usually develops unexpectedly after a wide yawn or opening the mouth wide for a bite, such as when eating an apple. Dislocation can occur with the relaxation of sleep and often with the wide yawn resulting when an individual wakes up. Trauma, if associated, is usually that of epileptic seizure; however, other sources of external trauma can be responsible.

As has been noted many times in this chapter, motion within the temporomandibular joint is directed more by muscle balance than by the shape of the articular surfaces and by the ligaments. The importance of muscular action is emphasized in dislocation of the temporomandibular joint. Normal movement of the condyle in translation is often to the anterior surface of the articular eminence, and the condyle easily returns to its usual position on the posterior surface.¹⁰⁷ In a cephalometric study of 100 joints, it was found that the condyle could normally move to a position 3 mm in front of the center of the articular eminence and return normally.^{94, 100} The amount the condyle moves on the eminence varies among individuals. It may not reach the crest of the eminence on maximum opening, stop at the crest, or go beyond the crest. There does not seem to be a significant relationship of condylar movement in regard to the crest with temporomandibular pain-dysfunction.¹¹² Although a relatively loose articular capsule is necessary for anterior dislocation of the condyle, it is not necessarily a contributing factor. Most individuals with excessively loose temporomandibular joints do not have anterior dislocation of the condyle.

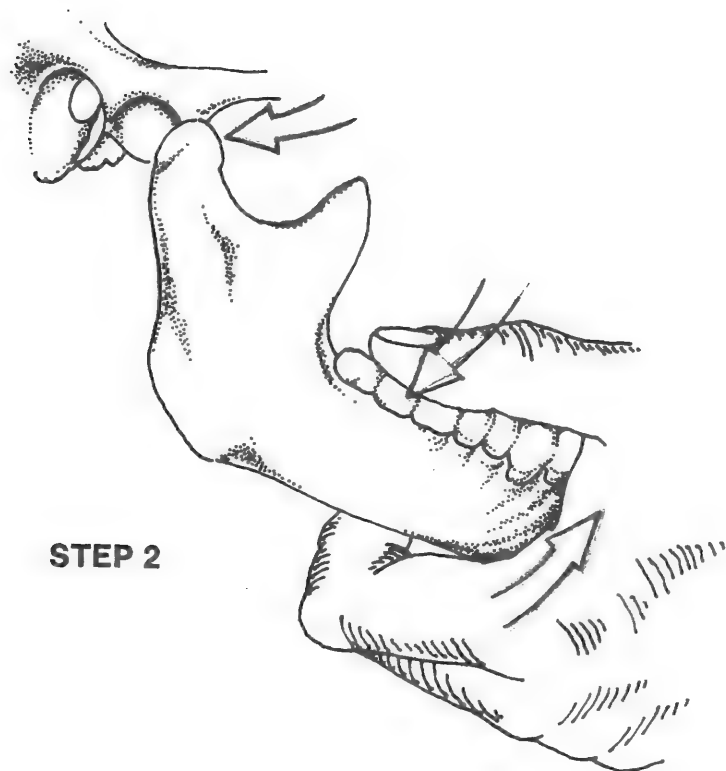
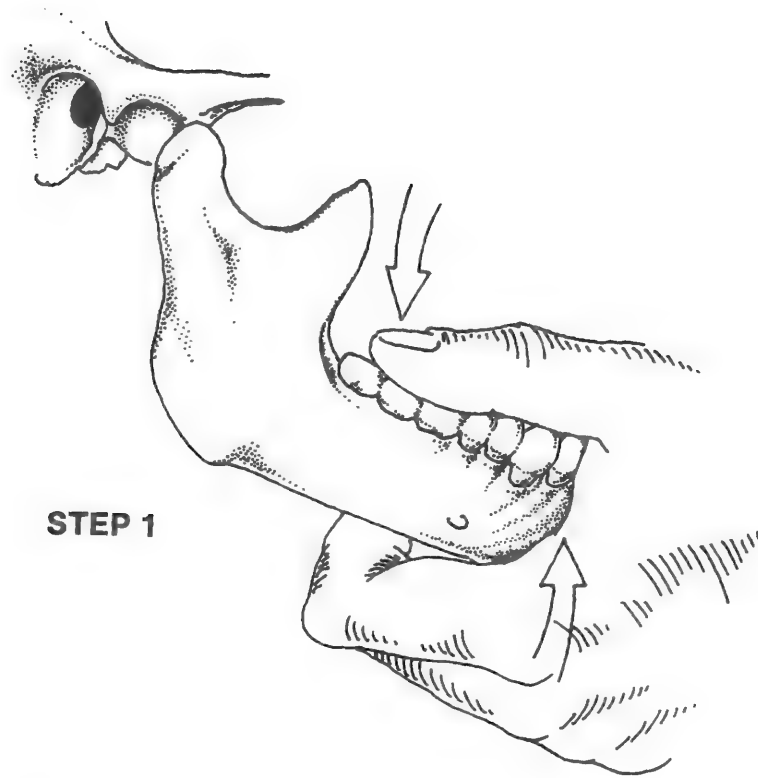
Dislocation often results from hypertonicity of the masseter and/or internal pterygoid muscles. When the condyle moves to the anterior surface of the articular eminence, contraction of these muscles does not allow it to return. This contraction is observed in electromyographic evaluation during biofeedback training for correction of the problem.⁴² On the completion of maximal opening, the jaw-closing muscles develop activity.⁶⁵ This appears to be a protective mechanism which is present in the normal individual. Applied kinesiology experience shows that when this contraction is excessive, there is a disturbance somewhere within the system interfering with normal control of muscle synergism. To bring the condyle back over the eminence, all

muscles must be functioning harmoniously, which includes activity of the posterior temporalis to draw the condyle back into the fossa.

The condyle can usually be easily manipulated into a normal position.¹³⁹ The physician's contact is much the same as for manipulating a disc. The physician contacts the most posterior molars with his thumb, and the fingers cradle the lower anterior aspect of the mandible. Gentle inferior pressure is applied to the molar teeth to distract the condyle inferiorly and stretch the masseter and internal pterygoid muscles. When relaxation takes place, the condyle is slipped over the articular eminence into the fossa. Duffy²³ pointed out that if the patient swallows just prior to the physician applying the posterior force, the translation of the condyle posteriorly over the eminence is much easier. The reason for the improvement of the manipulation with the swallowing maneuver is not clear, but it does significantly help. It is often necessary to have an assistant keep the patient's head from rotating away from the manipulative force to prevent loss of the manipulative action.

Dislocation of both sides at one time is rare; when this does occur, each side should be manipulated individually. The manipulation is done slowly, with calm direction. The patient is often in severe pain and fearful of the manipulative effort, although there is usually minimal discomfort associated with it and great relief immediately afterward. In some cases when the masseter and internal pterygoid are severely spasmed, it may be helpful to gently manipulate the neuromuscular spindle cells for relaxation. Ethyl chloride or Fluori-Methane spray may be helpful in relieving the muscle spasm and controlling pain.¹⁰⁶

Immediately following reduction of the TMJ dislocation, the muscles of the stomatognathic system, as well as of the cranium and occlusion, should be evaluated. The basic regime described on page 333 prior to the manipulation for the TMJ disc is applicable here, only in this instance the examination and treatment are applied after the joint manipulation. Additional consideration should be given to the internal pterygoid and masseter for muscle length and hypertonicity. Usual treatment is fascial release and trigger point techniques. Recurrent TMJ dislocation indicates that some factor in the stomatognathic system has not been corrected. Surgery to reduce the size of the articular eminence is not recommended. Patients who have been seen in applied kinesiology following this surgery have other types of temporomandibular joint problems and disturbance in the total stomatognathic system. Correction of these problems in the first place would probably have prevented the surgery.



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Chapter 12

Occlusion

In many places in this text we have already discussed the importance of the stomatognathic area with the stomatognathic system, and its relationship with total body function. Any study of muscular interactions within these divisions quickly reveals how only one area of imbalance can adversely affect other areas. Those in applied kinesiology in the earlier days did a considerable amount of work to balance the cervical flexors and extensors with the rest of the body. It was only a matter of time before it became necessary to evaluate and ultimately treat other muscles in the closed kinematic chain of the stomatognathic system, because it is usually impossible to effectively treat only portions of the system. Failure to correct one area will often cause loss of or poor corrections in other areas. Treatment to a localized area in the system may relieve the symptoms being treated, only to have new symptoms develop some place else within the complex. Often the patient and physician do not associate the two symptoms, considering them as individual entities. Fortunately, an increased awareness of body interactivities is developing.¹²³

With increased applied kinesiology understanding of the integration within this system, various divisions of the system were integrated into AK examination and treatment techniques. First the muscles of mastication were studied, followed quickly by the hyoid muscles. The methods for evaluating and balancing these muscle groups as presented by Goodheart^{45, 46} improved efforts to balance the cervical postural muscles and their organization with the rest of the body. Obviously, the next step of

interest to the applied kinesiologist was the dental occlusion itself. At that time very few physicians practicing applied kinesiology were dentally oriented; they had little, if any, knowledge about the importance of occlusion and its relation to the stomatognathic system, let alone to total body function. As more and more dentists became interested in applied kinesiology muscle balancing techniques, they began to introduce into AK their levels of expertise in evaluating and correcting malocclusion. This exchange of expertise on an interprofessional level has significantly improved the therapeutic approach for many patients. Not only have patients benefitted, but the physicians themselves have profited by being able to provide better health care.

As a result of understanding occlusal problems, the chiropractor and others using applied kinesiology methods can see why some conditions of the cranial primary respiratory mechanism and imbalances of postural muscles have not adequately held corrections. The dentist, on the other hand, better understands how occlusal corrections can improve function remote from the stomatognathic area, and also why specific types of symptomatic complaints are registered by patients after certain dental procedures. Previously these complaints were thought to be coincidental with — not necessarily related to — the procedure performed. The muscle examination and balancing techniques of applied kinesiology have enhanced the ability to obtain muscular balance prior to equilibration. AK techniques have also enabled easy evaluation of the cranial primary respiratory mechanism. If cranial faults are present,

Occlusion

there is a likelihood that the cranial base may be imbalanced. This must be corrected before proper equilibration can be accomplished.

This chapter, although entitled "Occlusion," certainly does not parallel the many excellent texts, workbooks, and monographs on the subject the dental profession has produced. The goal here is to discuss observations made in applied kinesiology regarding how occlusion influences the rest of the stomatognathic system, and also how we perceive that the total body influences occlusion. Gelb,⁴¹ reflecting on how a short leg adversely influences the dental occlusion, states, "Once again the unity of the body mechanism becomes apparent, with the stress of posture extending all the way up to the masticatory muscles, not down as we usually see it from a dental viewpoint."

To improve interprofessional communication, it is necessary for a physician who is not a dentist to better understand the problems a dentist faces in

dealing with malocclusion, and how intricate a process equilibration is. A dentist needs to be aware of how occlusal changes influence other areas of the body, both positively and negatively. At the least it is hoped that this chapter and others in this text will enable any doctor in the healing arts to do a screening examination in areas not within his particular field so that appropriate referral can be made for the patients' optimum treatment.

First it is necessary to understand what is meant by an optimum occlusion. It would be of no benefit here to define an optimum occlusion; it seems every text, workbook, and monograph has its own definition, probably because there have been many viewpoints on occlusion over the years. Currently there are many conflicting approaches for gaining an "optimum occlusion." It seems best to list the characteristics of an optimum occlusion, look at the factors which interfere with it, and see how malocclusion interferes with body function.

Optimum Occlusion

It is not always possible to obtain an optimum occlusion. To attain one goal it is sometimes necessary to violate another principle of occlusion. This is done when one factor is more important to total stomatognathic function. In some cases, bite planes, reconstructions, or partial or total prostheses may be necessary. In this discussion we consider how the occlusion influences the teeth, jaws, and — in a broader aspect — the total stomatognathic system and body itself. The factors listed as contributing to an optimum occlusion are discussed throughout this chapter.

1. Centric occlusion should be coincident with physiologic centric relation.

2. It is permissible and desirable to have a small amount of anterior superior slide from the terminal hinge axis (most retruded superior position of the condyle) to centric occlusion.

3. There should be no lateral deflective contacts, that is, a hit-and-slide from physiologic centric relation deflecting the mandible in a mesial or distal (lateral) direction.

4. There should be axial loading of the teeth, which means all forces should be directed in line with the long axes of the teeth.

5. There should be disocclusion, which is separation of teeth to prevent locking the occlusion in any eccentric movement.

6. Vertical dimension should be balanced with the physiologic rest position. This places the mandible in such a position that the temporomandibular joint is not compromised, nor is the freeway space.

7. Functional mastication should be provided. This considers the morphology of the tooth with its cusps and grooves to provide efficient cutting, tearing, and compression for chewing.

8. The occlusion and function of the jaws must be comfortable to the patient.

These factors of an optimum occlusion seem to satisfy the three basic biological laws which Ricketts⁹⁹ states as biologic standards. These are "... 1) the law of conservation of tissue, 2) the law of conservation of energy, and 3) the law of profound efficiency and maximum longevity."

There is considerable controversy as to where centric relation should be located. Since the goal of equilibration is to make centric relation and centric occlusion coincide, it is important to consider this controversy. The subject was discussed in the previous chapter. Review of some of the primary

thoughts leads us logically to the conclusion that optimum position for centric relation is with the condyle in the most superior position in the center of balanced muscle function.

When centric relation is considered the most retruded superior position of the condyle in the condylar fossa, the articulation is at its edge of motion.^{91, 92} This creates a problem because the joint cannot constantly adapt as necessary. As we will discuss, the occlusion is not necessarily stable. The apparent base for occlusion, the cranium itself, may shift position, changing the occlusion. Studies of the temporomandibular joint and dental arches indicate there is constant remodeling of the bone to meet the needs of adaptation.^{9, 10, 81, 82} When a joint functions at its edge of motion, there is no room for adaptation to the constantly changing status of physiology, morphology, and function. The position is one which is strained; no other joint in the body works at this edge of function.

Several studies have shown that in 80%-90% of apparently normal individuals, there was an anterior slide from a retruded centric relation to centric occlusion.^{91, 101} If one accepts the idea that the body is a self-correcting, self-maintaining mechanism, it is difficult to believe that all these apparently normal individuals had malocclusion. This concept would have to be accepted if the hypothesis of centric

occlusion and centric relation in the most retruded superior position is believed to be correct. Most individuals who have been equilibrated to have centric occlusion coincide with centric relation in the most retruded superior position have a posterior slide into centric occlusion as observed on the Mandibular Kinesiograph. This shows that the engram of closing is not adequately causing the muscles to have an habitual closing path to adapt to this man-made retruded position.

Additional evidence that the most retruded superior position is not optimum for centric relation is provided by the morphology of the temporomandibular joint. This position is not the strongest portion of the mandibular fossa for function.⁸⁷ The morphology of the disc is also disturbed by a most retruded position of the condyle in the fossa.³⁴ This position appears to compromise the posterior attachment of the disc, allowing the disc to move anteriorly and predisposing the joint to an opening click or opening lock during mandibular motion.

The ideal centric relation appears to be with the condyle in the most superior position, with its anterior to posterior relation in the center of balanced muscle function. This is termed "physiologic centric relation" in this text. The criteria necessary to obtain the physiologic centric relation is discussed further in the equilibration section of this chapter.

Etiology of Malocclusion

Malocclusion is simply "bad occlusion." It does not relate just to the teeth fitting together improperly in intercuspation; it should also include abnormal closure of the jaws. The teeth may intercuspate perfectly, but if there is a problem in the muscular balance which closes the jaws, the mandible may be slightly or grossly malpositioned as the teeth come together. This causes a hit-and-slide into the intercuspation of centric occlusion. Malocclusion can be hereditary, from disturbance in development (either intrinsic or extrinsic), or caused by some factor after development is complete.

There are several factors which cause structural disfigurement that are out of the scope of this text. These are inherited or congenital anomalies, or developmental problems as a result of endocrine or other intrinsic problems. This type of problem often creates severe malocclusion, which may require surgery or orthodontic treatment, or both. Malocclu-

sion has been shown to be accompanied by muscular imbalance observed on electromyography.⁸³ Surgery and orthodontic treatments may be enhanced by myofunctional therapy or applied kinesiology examination and treatment. These muscle therapies may be applicable prior to, coincident with, or after orthodontic or surgical procedures.

In this section we are more interested in malocclusion which has an acquired etiology such as trauma, improper habit patterns, remote body dysfunction, instability of the teeth, or iatrogenic causes. Consideration of etiology should also include nutrition, because there is abundant evidence that nutritional deficiencies can cause malocclusion. It doesn't even require what is generally considered malnutrition to produce a detrimental effect. A leader in this investigation has been Weston Price.⁹³ In studying various native cultures where the people were on the local unrefined diet, he found broad,

Occlusion

well-formed dental arches with no caries or malocclusion. After the western diet was introduced into the culture, narrow arches, caries, and malocclusion became commonplace. It appears quite obvious that nutritional deficiencies contribute to malocclusion and poor arch development. As we progress with the etiology of malocclusion, it should be stressed that there is no one factor which causes all malocclusion; probably severe malocclusion has numerous factors contributing to its etiology.

It is well-established that the shape of bone depends upon the physical stresses applied to it. This is known as Wolff's law,^{149, 150} and it has several applications to malocclusion. An early proponent of malocclusion caused by physical stress to the face was Harvey Stallard.^{126, 127, 128} First trained as an ecologist and then as an orthodontist, Stallard considered the environment of the face as a possible etiology of malocclusion. His first effort was to ask parents about the sleeping posture of their children. Unable to obtain adequate information because of poor parental observation, he began visiting the children's homes to observe them while they slept. The result of his early study was to find a high correlation of crossbites, V-shaped dental arches, and other types of malocclusion associated with different sleeping postures which put pressure on the face and jaw. The child applied force to these areas with a hand or arm stuck under the face, face and jaw mashed into the pillow, hands curled under the mandible, and other positions which put force into the maxilla or deviated the mandible. Stallard correlated other external stresses as a cause of malocclusion, such as propping the chin on the hand with the elbow rested on a desk, and thumb- or finger-sucking. He also related nutritional deficiencies as a compounding factor to external stresses applied to the stomatognathic area. Stallard studied over 7,000 children and found from observing the skull in general that he could predict much about the occlusion. If a child had a flattened posterior or posterolateral skull, it indicated that the primary

sleeping position was on the back, thus avoiding external stress to the face and mandible. These children had better occlusion overall than those who had the narrowed face of a face-sleeper. The reason the back-lying position is an optimum one in early childhood is that the occiput ossifies much more rapidly than the facial area.¹⁴⁸

Stallard observed external pressures on the face by how the bulky appliances used in orthodontic treatment at that time affected the adjacent tissues of the cheeks and lips. When a consistent habit pattern applied pressure to the face, the appliance would cut into the buccal mucosa if it were small and distant from the teeth. If the appliance was close to the teeth, the ridge in the mucosa would be as deep as the diameter of the appliance. His studies indicated that a child would have to rest directly upon the appliance for more than four or five hours daily in order to produce the cut or groove in the mucosa. When this appeared, he would have the child change his habit pattern, either consciously or with a harness apparatus, to maintain a sleeping posture on the back. The marks in the mucosa would quickly leave. On completion of orthodontic treatment and removal of the appliances, he often observed a relapse to malocclusion shortly after an improper habit pattern was resumed.

Malocclusion is often present in the early stages of development. In a study of 6,772 children it was found that 50% of the children between birth and two years of age had malocclusion. The figure increased to 68% of children in their mid-teens.¹²⁸ This study was done in the 1920s, showing that malocclusion is certainly not a new entity with which to deal in the United States.

The external forces of improper sleeping and habit patterns, as well as the nutritional factors of malocclusion, caries, and hyperkinesis, have recently been presented in a book for parents entitled *Why Raise Ugly Kids?*²⁵⁸ This book should help present these ideas to those interested in rearing their children in the best way possible.

OCCLUSION AND STABILITY OF TEETH

REMODELING

Movement of the teeth within the arches can create malocclusion, or the teeth can be adaptively moved to help correct it. It seems that a stable but adaptable tooth position is an important criterion for the maintenance of normal occlusion. Tooth position is maintained by the equilibrium of forces applied in all directions.¹⁴⁶ To maintain this equilibrium, there are occasions when it is necessary for the alveolar bone to remodel to allow a shift in tooth position for natural adaptive processes. This movement is limited and is generally gradual, adapting to the slow changes taking place in occlusion over a lifetime.³⁵ When the morphology of a tooth is changed by selective grinding to gain improved occlusion, the tooth will often shift slightly as a result of relieving the stress placed upon it by malocclusion.²⁴ Just as a tooth moves when stress is relieved, it also moves when stress is continually applied by malocclusion.¹⁴² This movement may cause adaptive remodeling of the alveolar bone, which results in remodeling of the tooth's position. This takes place by a rearrangement of the periodontal ligament fibers and resorption of the bone under pressure and consequent reconstruction of the bone,³¹ the same as when pressure is continually applied with orthodontic appliances for movement of teeth.

Several studies have been done at the University of Nebraska which reveal how the teeth are in constant equilibrium with forces placed upon them.¹⁴⁷ Attaway⁵ demonstrated that a force of as little as 1.6 gm can move a premolar tooth 0.033 inch, which is almost 1 mm, in eight weeks. Pressure was applied by the cheek as a result of modifying the first premolar tooth with a 2 mm gold inlay. A similar study was done by O'Meara.⁸⁸ This relatively small amount of pressure, combined with the considerable amount of movement, reveals how occlusion can be modified either favorably or adversely by the pressures applied by muscles and by how the occlusal surfaces contact during swallowing, etc.

There are many ways that muscular imbalance can cause movement of the teeth within the arches. Brodie¹² points out the importance of muscular balance in the development of the alveolar bone and eruption of the teeth. He calls the tongue and buccinator muscular envelope around the dental arches the "... first orthodontic forces to operate on the teeth and they continue to influence them profoundly throughout life." Examples are seen of muscle influencing bone and the occlusion in laboratory animals where muscles have been surgically

altered, resulting in an open bite, crossbite relationship, and atrophy of the tooth-bearing bone on the cut side.⁵⁵ As we will see, there are many ways that muscular imbalance can influence tooth position. In this light we see other examples of the statement, "When muscle and bone fight, muscle never loses." In addition to muscle balance, occlusal forces must be included in the balance of forces influencing tooth position.^{13, 49}

One way muscular imbalance can cause a shift in teeth is when the muscles cause malocclusion. This represents a combination of muscular and occlusal forces influencing the position of the teeth. The most powerful muscles influencing occlusion are the jaw closers, which may shift the mandible and cause the teeth to occlude improperly.⁷² Two reactions take place. The nervous system immediately attempts to change the muscular pattern because of stimulation to the periodontal ligament proprioceptors. If the muscle spasm or weakness causing the imbalance is predominant, the muscular system cannot adapt to the new neurologic pattern, and the malocclusion continues. This can occur when there has been damage to neuromuscular spindle cells or Golgi tendon organs within the muscle so that the muscle cannot respond to the appropriate neurological control, which is being governed by the stimulation of the periodontal ligament proprioceptors. Thus the first step of natural adaptation to malocclusion is not operable. The second reaction, a slower adaptive approach to malocclusion, begins when there is a persistency of malocclusion. A transient force as low as 0.5 gm is sufficient to displace a tooth on some individuals. In all individuals the amount of force needed for displacement is very small.⁷³ The absence of tooth equilibrium in its balance of forces may cause the alveolar bone to begin adaptive remodeling with a shift of tooth position. This normal reaction may eventually cause proper occlusion again if the desired tooth movement is not blocked by adjacent teeth. If proper occlusion develops and the muscular pattern remains disturbed, there will be equilibrium of forces. If the muscular imbalance which caused the malocclusion in the first place is corrected by the body's own therapeutic efforts or by physician intervention, malocclusion will develop in the opposite direction because of the remodeling which took place.

Muscular forces of the orofacial group, including the tongue, can also cause adaptive remodeling because of constant pressures applied to the teeth.⁵⁰ Major examples of this are the various types of tongue thrust which may be present during swallow-

Occlusion

ing or at rest. Constantly thrusting the tongue against the upper incisors during swallowing causes remodeling of the alveolar bone, with the incisor teeth going into labioversion. The problem is compounded if the orbicularis oris is weak and does not provide pressure counteracting the thrusting tongue.

Another type of malocclusion can develop as a result of holding the tongue between the molars during rest position and swallowing; this causes the molars to intrude, resulting in a posterior open bite. This, in turn, causes lack of posterior support and results in overclosure and stress to the temporomandibular joint. There are many types of orofacial muscular imbalances which can adversely influence the occlusion. These are discussed thoroughly in Chapter 15.

PERIODONTAL DISEASE

Periodontal disease and the resulting instability of the teeth affect the occlusion, and there is considerable evidence that malocclusion is a major factor in periodontal disease. There is some controversy about whether traumatic occlusion can be a primary factor in periodontal disease; some periodontists state that trauma cannot cause the condition until secondary bacterial invasion is present. As Ricketts⁹⁹ points out, "Even with this explanation, trauma is a part of the equation."

In the late 1940s and early 1950s, much was written by periodontists concerning the effects of malocclusion on the periodontal tissues. Their interest in occlusion added much to the techniques of equilibration, and for a time they were the primary specialists who equilibrated natural dentition. Techniques of equilibration have now been more generally embraced by the broad field of dentistry.

Mental stress appears to be a factor in the development of periodontal disease,^{23, 26} probably for two reasons. (1) Stress increases bruxing^{107, 109, 151} with resulting trauma to the periodontal tissues, probably of greater magnitude than that caused by malocclusion. (2) Stress also appears to correlate with a metabolic factor, increasing the incidence of periodontal disease.¹¹⁵ In several correlated studies, Cheraskin, Ringsdorf et al.^{14, 15, 16, 17, 18, 19, 20, 21, 102, 103} demonstrated a detrimental effect on oral health from short-term increased refined carbohydrate intake. There was a statistically significant increase of gingival inflammation, pocket depth, and tooth mobility coincident with an abnormal change in the electrocardiogram of a depressed T wave on lead I.^{20, 139} Another study by them showed a reduced sulcus depth after only four days of reduced carbohydrate intake.^{102, 103} Selye¹¹⁵ observed that stress (General Adaptation Syndrome) "... no

doubt exert(s) an important influence on the development and maintenance of dental structures." Sugar and stress being detrimental to oral health parallel the applied kinesiology observation that refined carbohydrate intake increases general stress in the body. (Blood sugar regulation and stress are considered in Volume V.)

Vitamin C deficiency, with its associated negative fiber and bone balance, will lead to a failure of the periodontal transmission mechanism.³⁰ This will ultimately lead to periodontal disease and possibly loss of teeth.

Any activity which continually causes trauma to the periodontal tissues can cause periodontal disease. Habits are very frequently the etiology of the problem.¹²⁵ Among these may be pipe smoking, toothpick chewing, bracing with emotional stress, etc. There are also occupational habits, such as holding nails between the teeth, cutting thread, etc.

Folic acid apparently has the effect on the gingiva of increasing resistance to local irritants and leading to reduction in inflammation of the periodontal tissues. A double-blind study by Vogel et al.¹⁴¹ produced a significant reduction in gingival exudate flow after thirty days of ingesting 2 mg of crystalline folic acid twice daily. There was no improvement in the gingival index; however, exudate flow is considered an early indicator of changes in gingival inflammation.³³ (The overall viewpoint of applied kinesiology on folic acid is discussed in Volume V.)

A general nutritional evaluation of a patient with periodontal disturbance is necessary. Proper nutritional support and dietary corrections should be instituted. Gelb⁴¹ finds supplementation with cold processed raw veal bone which contains the nucleoprotein extract and natural enzymes to be of value in the degenerative conditions associated with disturbances of the stomatognathic system. His observations parallel those of applied kinesiology for this supplement; both are clinical observations.

CRANIAL INFLUENCE ON STRUCTURE

It has already been observed in Stallard's studies that cranial and facial balance can be disturbed by external pressures applied on a consistent basis, molding the bone according to Wolff's law. The skull may also become structurally imbalanced as a result of its inherent mobility, discussed in the first section of this text. This can cause a distorted base from which the mandible functions. The resulting malocclusion may be gross or relatively minimal. The amount of malocclusion does not necessarily indicate the number and severity of symptoms which might develop from the problem.

Cranial imbalance may develop from the time of

birth throughout life. Many problems of cranial balance and function develop from birth trauma, when the skull is distorted during the birth process and does not regain balance; it may ultimately ossify in a structurally distorted manner. Studies have indicated that a difficult birth has a cause-effect relation with dysfunction of the cranial-sacral primary respiratory system.^{36, 140} At a minimum, this appears to interfere with the normal nerve function necessary to control the balance of the muscles of the stomatognathic system. This type of cranial disturbance influencing occlusion will be discussed later.

As stated, cranial imbalance — whether from external pressure factors as discussed above or from birth trauma — creates an imbalanced base from which the mandible and its muscular attachments function. The usual concept is that the cranium is the solid base of occlusion. In light of current knowledge, this concept of occlusion must be modified to include the dynamic mobile complex that the skull has been shown to be.^{6, 37, 79, 96, 132, 134} Malocclusion can develop as a result of the temporal bones counter-rotating with one another.⁷⁵ This changes the relation of the mandibular fossae and consequently the mandibular condyles, ultimately changing the position of the mandible in its relation to the upper arch. This shift is more severe if an individual has been equilibrated to centric occlusion coincident with centric relation in a most retruded superior position. If centric occlusion is coincident with physiologic centric relation, there is a greater ability to adapt since function is not at the edge of the envelope of motion. Another way improper cranial primary respiratory function mechanically alters the occlusion is movement of the maxillae, changing the shape of the maxillary arch. This has been shown to occur from cranial treatment⁶ and can just as easily develop as a result of trauma to the skull.

An imbalanced skull, whether from birth trauma, external pressures, or any one of the other causes of cranial faults, does not necessarily mean there will be a malocclusion. The body is a self-correcting, self-maintaining mechanism, and normal adaptation develops according to the circumstances. This is accomplished by remodeling processes of bone. The alveolar process may remodel to change tooth position. The condyle and mandibular fossa change to accommodate each other.^{9, 10, 81} There is also neuromuscular control which causes the muscles of mastication to act in proper synergism to close the mandible into proper intercuspation, avoiding premature tooth contacts. This unerring closure produces a solid "clack" sound^{122, 129} and is the result of a proper engram having developed because of proprioceptive communication from the periodontal liga-

ment, mucous membranes, gingiva, and temporomandibular joints.

Whether normal occlusion is because of adaptation to an abnormal cranial balance or complete normalcy of the cranium and occlusion, any change in cranial function will disturb the occlusion. As an example, let's consider an imbalanced skull. It makes no difference whether the skull is distorted from birth trauma, external forces such as sleeping or thumb-sucking, or any type of general trauma. The body attempts to develop occlusion in an optimum manner for the circumstances present. As a result, there may be balance in the closed kinematic chain and no improper signaling in the afferent system. Introduce into the picture a physician familiar with cranial primary respiratory function; he observes digestive disturbance from cranial nerve dysfunction, resulting from the cranial stress present. The physician proceeds to make cranial corrections which, although proper for the cranial primary respiratory mechanism, interfere with the developed adaptive occlusion. The upper dental arch changes as the maxillae gain a different position with each other. The temporal bones are brought into balance, changing the mandibular fossae relationship and consequently mandibular position. This rapid change of the jaws' relation with each other, and even the relation of the teeth within the upper arch, is not one which can be quickly accommodated by adaptation. If the cranial position is changed slowly over a prolonged period, there will be remodeling of the bone to make the adaptation. With the current procedures of cranial correction, the change of skull position is not that slow.

Under these circumstances, one of two reactions will take place within the system. As soon as an individual chews or bites down, the established occlusion will immediately force the cranial bones back to their position prior to correction. There is then no change from the health status prior to the corrective intervention in the cranial primary respiratory mechanism. On the other hand, if the cranial correction is dominant, there is now malocclusion which will be recognized by the afferent nervous system; it will immediately begin to build a new engram of muscle closure to eliminate the prematurities which have developed as a result of the change. The adaptation of an engram to a new status of occlusion is quite rapid.⁹² If the change needed by the nervous system to bring the jaw closers into a synergistic harmony to eliminate the malocclusion is minimal, it will be accomplished. In other words, physiologic centric relation and occlusion will become coincident. If the change of occlusion brings about too many prematurities, especially those of conflicting

Occlusion

nature where one indicates the mandible should move slightly to the right and another indicates to the left, confusion develops within the nervous system. This will possibly result in a massive imbalance in the closed kinematic chain of the stomatognathic system, ultimately affecting the entire body. In this case, if the cranial correction continues to hold, the only possibility for natural adaptation is remodeling of the tooth position which the body may or may not be able to accomplish. This is an example of what can take place when there are cranial faults with normal occlusion and the faults are corrected. Bringing the cranium into normal function may create malocclusion.

Another example of cranial structure relating with occlusion begins with normal cranial function and structure coincident with normal arch formation and occlusion. A blow to the head or any other etiology of cranial disturbance can create malocclusion, again by changing the relationship of the temporal bones and thus the mandibular fossa, as well as changing the shape of the maxillary arch. Fortunately, the body is a self-correcting and self-maintaining mechanism. Often under these circumstances the activity of biting down and chewing corrects the cranial fault. The mandible is a single bone, so its arch acts as a relatively stable base as opposed to the maxillary arch which is made up of two bones. During biting, the teeth of the distorted maxillary arch are forced to intercusate with the teeth of the mandibular arch. This tends to form the maxillary arch to fit the mandibular arch; the strong muscles of mastication pull into the cranium, the forces of which tend to balance the previously unbalanced cranium to match the relatively solid base of the mandible. Correction obtained by this natural mechanism depends on which is dominant, the cranial fault or the previously correct occlusion. If the cranial fault is dominant there is now malocclusion and, as in the previous example, the afferent nervous system begins adaptation, if possible. If it cannot, there will be confusion within the nervous system, and a problem similar to the previous example will develop.

There may be variations in the example of a normal cranium and occlusion disturbed by trauma. If a cranial fault develops and the person senses malocclusion, temporomandibular joint pain, or some other symptom, a dentist may be consulted. The dentist recognizes that the temporomandibular joint pain is caused by malocclusion. Treatment may be bite plane therapy, followed by equilibration to relieve the muscular spasms and stress on the teeth. This often eliminates the symptomatic pattern for which the patient sought help. Now a new problem is

present which may never be related with the recent procedures. The mandibular arch, a relatively stable base in occlusion, has been made to match the skull's position when cranial faults are present, causing the cranial faults to be locked in. They may, by way of peripheral entrapment of the cranial nerves or by other mechanisms, cause dysfunction and symptoms remote from the stomatognathic system. Unless a physician familiar with the integration of these factors becomes involved in the case, the person will probably receive treatment for the secondary problems with no attention given to the primary cause.

The main emphasis here is to point out that the cranium is not the stable base of occlusion that most consider it to be. Several dentists have pointed this out in the literature;^{6, 42, 52, 53, 122} however, it is not a well-known and generally practiced concept. This is unfortunate, because improper interaction of the cranial primary respiratory mechanism with occlusion is the reason many cases fail to stabilize even though excellent procedures of equilibration are used.

MUSCULAR BALANCE

Normal occlusion is a product of many structural considerations and physiological actions. Electromyography shows that each individual develops his own synergistic muscular action for an individual closing pattern.⁸⁴ An extremely important piece of the total picture is proprioceptive guidance of the jaw-closing muscles to unerringly and unhesitatingly bring the jaws into intercusation, with no premature contact causing a resultant slide into the final position. The proprioceptive mechanism providing the information for an engram to control the muscles can be compared with other body activities. The only difference between the engram in the masticatory system and others in the body is the accuracy with which it functions.

A similar type of engram is responsible for enabling an individual to close his eyes and swing his arm in a wide circle, bringing a finger unerringly to touch his nose. This is an engram of learned muscular response. Any engram must be continually reinforced by repeated stimulation of the proprioceptors, or its accuracy is progressively lost. The engram of a golf swing, once learned, will always be present; however, without practice and re-stimulation of the proprioceptors in the engram pattern, precision of movement will be lost. The nicety of the mandibular closing engram can be recognized when its accuracy is considered in view of the fact that mandibular closing is almost totally regulated by muscles, and less by the shape of the articulating bones and ligaments than any other joint in the body.¹¹⁴

The engram is built primarily from the periodontal proprioceptors which, according to Shore,¹¹⁸ provide 90% of the proprioception, with the remaining 10% coming from the temporomandibular joint. Although not designated as such by Shore, there is probably some proprioception coming from the gingiva and mucous membranes of the oral cavity. When teeth are lost, the vast majority of proprioception for jaw closing is also lost; consequently, there is a loss of closing guidance in edentulous patients.³¹ In this situation, guidance is by the limited proprioceptors in the temporomandibular joint; there is additional proprioceptive information from pressure on the mucoperiosteum of the toothless alveolar ridges by food or a denture. It is unknown whether sensory information from this tissue can help guide the jaw closers. There seems to be some correlation with the original proprioceptive function of the periodontal ligament. Clinical evidence of this is seen in the correlation of the edentulous ridge with the homuncular representation of the teeth which has been observed in applied kinesiology (see Chapter 9).

The engram of mandibular closing as described so far is already a very integrated neuromuscular function. Detail has been presented regarding how the masticatory muscles must work in a synergistic manner to perform the habitual arc of closing with unerring accuracy. This is probably pointed out best by the moderately effective function of the mandible when the condyle is absent congenitally or has been lost through condylectomy.^{54, 67}

Consideration of the highly developed neuromuscular interaction must be taken a step further to understand the total complex. The muscles of mastication are only a part of the closed muscular kinematic chain. When the neck and head are nodded forward by the relaxation of the extensors, the antagonistic action of the hyoid muscles changes. Nodding the head forward can be a normal postural movement, or it can be caused by weakness of the neck extensors which can result from many causes. Whether the head is nodded forward from a normal postural change or abnormally from muscle weakness, there must be change in the neuromuscular pattern of jaw closing to maintain the unerring movement of the mandible to centric occlusion. This makes it necessary to consider all structures and functions which have an influence on mandibular movement and, finally, the occlusion itself. The interaction and adaptation of the engram become even more complex if the cervical flexion is with rotation and head tilt. In this case, one side of the hyoid muscles elongates and the other shortens.

Lieb⁷⁴ looks at this interaction from the dental viewpoint. * *"We are being forced to recognize more*

and more that occlusion of the teeth is not the be-all and end-all of our endeavors. As we find that many of these problems arise more from abnormal tonicity of the muscles involved, we are forced to think of occlusion as more than a teeth-to-teeth relationship. We see now that mal-relationship of the jaws has a consequent effect on the whole neuromuscular system involved in the various static and active functions of the structures in the head, neck, and shoulders of our patients. Occlusion now comes to mean the relationship of all the parts of the dental apparatus: the jaws, muscles, joints, and all the surrounding structures, not the teeth alone. We must consider the relationship of the dental apparatus to its surroundings and investigate further the functions of the various parts. The relationship of structure is, of course, a structural concept, and, since posture and balance are orthopedic in nature, the term 'oral orthopedics' was selected as descriptive of the entire discipline."

This muscular interaction occurs throughout the body and is a two-way street. It is possible for malocclusion to cause an imbalance in the masticatory muscles, which in turn disturbs the closed kinematic chain of the stomatognathic system and changes posture, ultimately causing low back pain and many other problems. Conversely, a disturbance in posture can interfere with balance in the closed kinematic chain in the stomatognathic system, ultimately causing malocclusion. These interactions must be precisely understood by the various disciplines in the healing arts, because the ultimate area needing treatment may require consultation with and treatment by a physician in another discipline. Failure to recognize the basic underlying cause of a problem results in an unresponsive patient; it can sometimes even cause iatrogenic problems because of treatment in the wrong area.

In considering the integration of the muscles of mastication and jaws with body structure, it must be remembered how important the muscles' activity is to the cranial primary respiratory mechanism. This has already been pointed out in several areas of this text. The two-way interaction of the muscles of mastication and the cranium should be kept uppermost in one's mind as the neuromuscular complex regulating mandibular movement is discussed.

Additional considerations of mandibular activity are necessary before the total interaction of this complex can be considered complete. The jaws are usually thought of as the mechanism for ingesting

*From Myron E. Lieb, "Oral Orthopedics," in *Clinical Management of Head, Neck and TMJ Pain and Dysfunction*, ed. Harold Gelb (Philadelphia: W.B. Saunders Co., 1977), with permission.

Occlusion

food by chewing and swallowing. The additional activities of speaking and breathing must be considered.⁹⁸ The mouth acts as a secondary organ of respiration. If an individual is a chronic mouth breather, there will be an influence on the total organization of the system. The activities of speech require additional muscular engrams of both the muscles of mastication and the orofacial group, as well as those of the hyoid. Breathing or speech disturbances may change the rest position and should be considered when it is measured.⁹⁷ In some cases dysfunction of breathing or speaking can be significant in causing a disturbance in the total system. As with other areas of primary involvement, they may easily be overlooked; thus treatment may be provided to an improper aspect.

There are many ways that muscles can cause malocclusion. Consider the complexity and many areas of integration evidenced by the engram of closing, developed from periodontal ligament proprioception. There is interaction within the closed kinematic chain which receives outside influences from throughout the body. Activities such as speaking, breathing, and deglutition are modifications of the closing engram. Improper function of any of these neuromuscular activities can ultimately manifest as malocclusion.

The most common and direct muscular imbalance is improper function of the muscles of mastication themselves. This appears to be primarily from structural problems within the muscles, or from psychological factors. A psychological factor manifests as bracing or bruxism,^{72, 107, 108, 109} causing persistent or recurrent hyperactivity of the muscles which can ultimately cause tooth movement as described previously. Care must be taken in differential diagnosis that emotionally-induced hypertonicity is the total problem before the physician assigns the etiology of malocclusion to this cause. Often clenching, bracing, and bruxism are secondary to other factors not totally related with emotional stress. (The applied kinesiology approach to emotional stress is discussed in Volume V.)

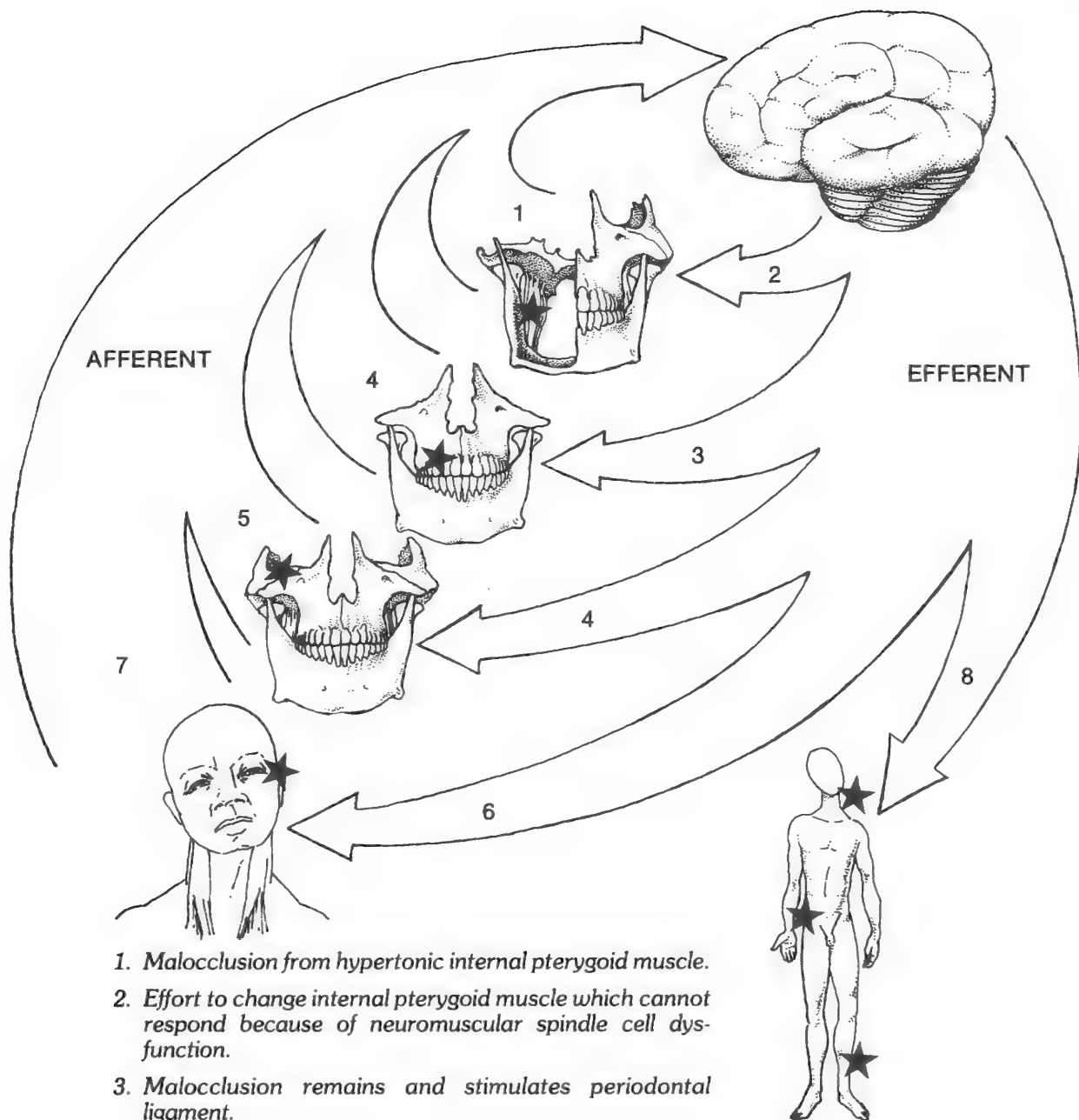
Direct muscular imbalance is generally associated with dysfunction of the neuromuscular spindle cell, and occasionally the Golgi tendon organ.⁴⁵ Failure of these muscle proprioceptors to function properly is usually due to trauma which may be induced by overstretching or overcontracting the muscle; rarely is it caused by trauma directly to the proprioceptor. It appears that trauma to the neuromuscular spindle cell can cause adhesions in the delicate septa between the intra- and extrafusal fibers, swelling within the spindle, or direct trauma to the nuclear bag area.¹⁴³ This can cause afferent stimulation from

the spindle cell to be either over- or underactive, resulting in hypertonicity or weakness in the homonymous, synergistic, or fixator muscles associated with it. It can also improperly stimulate the associated antagonist muscles.

What appears to be simple dysfunction of a neuromuscular spindle cell can be the primary cause of an entire stomatognathic system imbalance. Ramifications of afferent stimulation from the dysfunctioning neuromuscular spindle cell have very widespread effects.

An example of neuromuscular spindle cell dysfunction in an internal pterygoid muscle illustrates the many dysfunctions which can develop. If the result of improper afferent stimulation is hypertonicity of the internal pterygoid muscle, the normal engram of mandibular closing is overridden and the mandible is off-center, creating prematurities as the teeth come into centric occlusion. Let's hypothetically follow what may take place as a result of this malocclusion. First, as a result of the stimulation to the periodontal ligament proprioceptors from the prematurity, the engram of closing begins to change. There would probably be stimulation of the gamma efferent to the internal pterygoid muscle to effect relaxation. Because the neuromuscular spindle cell is not functioning normally, the muscle fails to respond. This status will be maintained until the noxious afferent impulse coming from the traumatized neuromuscular spindle cell is spontaneously removed, or appropriate therapeutic actions are applied to return it to normal. Often the condition is self-limiting, with the swelling of the neuromuscular spindle cell being resorbed or the nuclear bag fibers repairing. If trauma to the spindle cell results in adhesions between the intra- and extrafusal fibers, it appears necessary to therapeutically break these adhesions so that there can be independent action of the intra- and extrafusal fibers. In any event, the malocclusion and its influence on the stomatognathic system, which in turn may influence the total body, remain until the neuromuscular spindle cell is returned to normal.

Neuromuscular imbalance resulting from disturbance in the neuromuscular spindle cell of the internal pterygoid muscle is not limited to the occlusal change described above. The neuromuscular spindle cell within a muscle facilitates synergistic and fixator muscles and inhibits antagonistic muscles. Because the internal pterygoid muscle is part of the closed kinematic chain of the stomatognathic system, it can influence the neck flexors or extensors and hyoid muscles, as well as the muscles of mastication. As has already been pointed out, when the closed kinematic chain of the stomatognathic system be-



12—1. Schematic representation of possible effects from malocclusion.

Occlusion

comes imbalanced, it may affect postural balance throughout the body; structural strain, vertebral subluxations, and numerous other problems may develop.

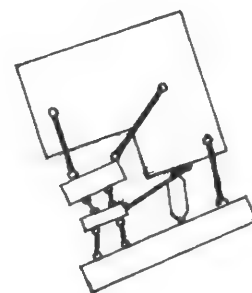
Obviously, similar reactions can develop when the malfunctioning proprioceptor is located in any of the muscles of mastication, ultimately affecting the closed kinematic chain of the stomatognathic system. But what about proprioceptors located remotely in the body ultimately affecting the occlusion? Let's use an example as structurally remote from the occlusion as possible. If there is a structural fault in the foot, such as pronation, the joint and muscle proprioceptors active in the positive support mechanism to facilitate the extensor muscles and aid the body in maintaining an upright position may, instead of facilitating neck extensors, cause inhibition. (These structural problems are discussed thoroughly in Volumes I and IV). Thus the closed kinematic chain of the stomatognathic system becomes imbalanced, and a chain of events reverse to that described above develops. Because of the relaxation of the neck

extensor muscles, the hyoid group is influenced and usually contracted. Since these muscles must relax in the proper manner on mandibular closing, the pattern of the habitual arc controlled by the closing engram is disturbed. If the inhibition of the neck extensors is unilateral, with secondary facilitation of the hyoid muscles, there will be more change to the mandibular movement than if the problem is bilateral. The malocclusion will probably be manifested as a lateral slide; with this stimulation to the periodontal proprioceptors, additional muscular imbalances of the stomatognathic system will probably develop.

The primary muscle proprioceptive dysfunction may be located almost anywhere in the body. It is necessary to have a systematic method of examination to efficiently locate dysfunction so that correction can be quickly and effectively obtained. The methods for correction of the stomatognathic system are described in Chapters 13-17 of this text. Procedures for examination and treatment of proprioceptors outside the stomatognathic system are described in Volumes I and IV.



Step 3. Closed kinematic chain of the stomatognathic system becomes imbalanced, changing the occlusion.



Step 2. Failure of extensor muscles to be facilitated by positive support mechanism.

Step 1. Improper stimulation of the positive support nerve receptors in the foot because of a foot subluxation.

12—2. Chain of events which may cause malocclusion as a result of remote dysfunction (see page 280-281 for description of the closed kinematic chain as illustrated by block diagram).

Trigger Points

Travell^{137, 138} has described trigger points associated with the sternocleidomastoid, upper trapezius, and the muscles of mastication. These are usually related with referred pain. In applied kinesiology it has been demonstrated that the muscle involved with the trigger point becomes weak immediately after it is stretched.^{47, 143} This is called the muscle stretch reaction; it helps give a diagnostic indication of the presence of trigger points. Muscles so involved are short, but it is questionable whether they are hypertonic. In any event, when trigger points as described by Travell are present, there is muscular imbalance which can cause malocclusion and adversely influence the total stomatognathic system as well as the rest of the body.

Fascial Release

Rolf¹⁰⁶ describes a muscular condition where the muscle and its fascia do not function harmoniously. When this condition is present, it can also be identified by the muscle stretch reaction. These muscles are shortened but, like those with trigger points, they may not be hypertonic. Their influence on the stomatognathic system is similar to those with trigger points, but there is not the referred pain and neurologic aspects of the trigger point. These muscles are treated with a deep massage to break down and "iron out" the apparent adhesions between the muscle fibers and their fascia.

Cranial Nerve Control

Most of the muscles of the stomatognathic system are under the control of cranial nerves. The majority of the hyoid muscles receive their supply from cranial nerve XII, the muscles of mastication from cranial V, and the sternocleidomastoid and trapezius from cranial nerve XI. Cranial faults which create peripheral entrapment of the cranial nerves may influence the stomatognathic system, and ultimately the occlusion, in many ways. There can be direct disturbance of the masticatory muscles by entrapment of cranial V. The entire series of events from a resulting prematurity may take place, ultimately affecting the entire stomatognathic system and having an adverse influence on the entire body. The occlusion may be indirectly affected by imbalance of the hyoid muscles or head-balancing muscles, such as the sternocleidomastoid and upper trapezius. Before any change is done to the resulting malocclusion, the cranial primary respiratory mechanism should be evaluated and corrected, as described in Section I of this volume.

Spinal Subluxations

Muscular imbalance in the stomatognathic system can be the result of spinal subluxations. Direct influence on the closed kinematic chain of the stomatognathic system is not as frequently a result of disturbance in the lower spinal area as those previously discussed. There is frequently direct influence on the closed kinematic chain from the upper cervical area, which relates on an afferent basis with the sternocleidomastoid and upper trapezius muscles. Their imbalance can cause head-leveling problems which rapidly encompass the total stomatognathic system. Direct evidence of head position with malocclusion has been demonstrated electromyographically by Funakoshi et al.³⁹ This influence is from the tonic neck reflex,³⁸ which has its receptors in the ligaments of the upper cervical vertebrae.

Vertebral and pelvic subluxations can influence the stomatognathic system on a remote basis. The resulting structural distortion may create shoulder or head-leveling problems. The cranial mechanism may become secondarily involved, influencing the cranial nerves. Wide and varying complexes are possible from this type of dysfunction.

The muscular causes of malocclusion are many and varied. They can occur as a result of disturbance within the muscle itself, which ultimately affects other muscles; they may be a result of improper control of the muscle, such as in the peripheral nerve entrapment of cranial faults or vertebral subluxations. Nearly all approaches to malocclusion recommend that muscular imbalance be corrected prior to equilibration. The problem is that most approaches do not take into consideration the multiplicity of etiology concerning muscular imbalance. Some procedures correct the muscular imbalance more by accident than by design. These fall within the realm of physical and electrotherapy, which may return a muscle proprioceptor to normal function; unfortunately, the procedures usually require repeated application over a relatively long time period. Exact diagnosis and application of treatment by applied kinesiology techniques can immediately return muscle proprioceptors to normal function. Immediate return of the muscles to normal function is also obtained by cranial fault and vertebral subluxation correction. If muscle dysfunction returns, it is strong indication that treatment applied is to a secondary area rather than a primary area of fault. Continued evaluation is necessary to find the primary disturbance which, when corrected, will have a lasting effect.

VERTICAL DIMENSION

So far this discussion on the etiology of malocclusion has related only to the two dimensions of mandibular movement which are in the direction of the transverse or horizontal plane. As with every other structural consideration in the body, occlusion is three-dimensional. Vertical dimension, the third dimension of occlusion, is the measurement of the skull to the mandible. It can be considered as "... a vertical measurement of the face between any two arbitrarily selected points which are conveniently located, one above and one below the mouth, usually in the midline."²⁹ Generally when vertical dimension is discussed, it is in reference to the amount of freeway space between the upper and lower dental arches when the mandible is in its physiologic rest position. This is called the rest vertical dimension. Posselt⁹² refers to this as the postural vertical dimension. The term "postural position," which is synonymous with rest position, must be clearly understood when considering this very important dimension. It is defined by Posselt as "... the relation of the mandible to the maxilla with normal tonus of the masticatory muscles and with the subject standing or sitting upright." As was pointed out in the previous chapter, the rest position is dynamic with postural change. The rest position cannot be determined in the presence of any muscular imbalance. The most frequent muscle consideration is for hypertonicity. Muscles can also be hypotonic, or weak, which is more difficult to determine in the masticatory apparatus.

Occlusal vertical dimension is defined as "... the vertical dimension of the face with the teeth occluding in the intercuspal position."⁹² The difference between the occlusal vertical dimension and the physiologic rest vertical dimension is the freeway space. Maintaining normal freeway space is extremely important for harmonious function in the stomatognathic system. Some change in the vertical dimension is well-tolerated by the system, but if the change becomes too great disastrous results can develop.

The first consideration of vertical dimension is whether there was proper development in the relation of the cranial bones, facial bones, and mandible. Failure of mandibular development can cause an improper vertical dimension which may be severe enough to require surgical and/or orthodontic intervention. Some have espoused the philosophy that there has been an overall lack of development of the mandible in current generations because of malnutrition and genetics. This philosophy is proposed primarily by those who find a decreased vertical dimension in nearly everyone they examine.

Rather than consider that the function of the general population is abnormal, it seems more reasonable that the examination procedures used in this philosophy are not in keeping with the current status of function in the general population. Using the procedures of examination and correction presented in this text, one can see that many individuals classified as having a diminished vertical dimension by various examination procedures no longer have the indications after muscle balancing and other procedures have been completed.

Reduced Vertical Dimension

Excessive reduction of vertical dimension causes the condyle to move superiorly into the articular fossa during centric occlusion. This changes the dynamics of the temporomandibular joint, and also the relationship of the stomatognathic area with the rest of the stomatognathic system and the total body. The preponderance of evidence is that excessive loss of vertical dimension can adversely influence the joint and also the harmony of the total system. There is considerable controversy regarding the importance of loss of vertical dimension; this will be discussed later in the chapter under "Increasing Vertical Dimension."

A common cause of loss of posterior support is the loss of molar teeth. Since the stomatognathic system changes rapidly, symptoms develop almost immediately. Other causes of loss of vertical dimension are more insidious, and often the symptoms which develop may not seem to be related to the TMJ and dentition. Symptoms can range from headaches, visual disturbance, and auditory problems to symptoms throughout the body. The symptomatic picture is often treated without adequate consideration given to the basic underlying problem of malocclusion. Considering physicians of all disciplines, the dentist is most often the one who finds loss of vertical dimension as the primary cause of the symptoms. It is unfortunate that often a patient has first consulted many doctors who are not aware of this interaction; thus the symptomatic pattern is treated with limited or no results because the symptoms are so often remote from the basic underlying cause of the problem. A patient may seek treatment from an internist who recognizes disturbances resulting from cranial nerve dysfunction and uses medication to attempt to correct the problem. The symptoms may be skeletal and receive chiropractic attention. If the chiropractor is not aware of the interaction of the occlusion with the closed kinematic chain of the stomatognathic system, treatment may be directed to subluxations in the cervical spine which result from muscular imbalance.

Again, only a temporary symptomatic improvement will be obtained as a result of the therapeutic efforts. The techniques of applied kinesiology examination presented in Chapter 14 readily find the cause to be loss of vertical dimension if, in fact, it is present.

Insidious loss of vertical dimension is from wear of the teeth or their intrusion. Because this problem develops over a prolonged period, there is even less recognition by various physicians of the possibility that the remote symptoms are associated with malocclusion. When loss of teeth causes loss of posterior support, the symptoms often develop almost simultaneously with the tooth loss. This helps tie in the new symptoms with the change in dentition. This helpful diagnostic factor is not present in the insidious loss of vertical dimension.

Wear significant enough to reduce the vertical dimension is usually pathological. In normal mastication, the teeth do not come in contact; they are held slightly apart by the food.^{59, 60} This means that the teeth chew the food rather than chewing each other, which is particularly true with the soft food diet currently prevalent. An exception could be in cultures where the food has a high abrasive content.

An abnormal activity of the masticatory system is bruxism, which is gnashing and grinding of the teeth. Bruxism is defined as "... grinding of the teeth in other than chewing movements of the mandible, especially such movements performed during sleep."²⁸ It is primarily a horizontal movement which wears the cusps of the teeth, causing a flattening of the occlusal surface.

Counteracting the wear factor of the teeth as a cause of vertical dimension loss is the body's compensatory mechanism of continuous eruption.^{11, 51} The continuous eruption of teeth is held in check by occlusion or some other factor which provides a stop, such as the tongue or cheek being held between the teeth. To a certain extent there is a reserve of tooth eruption available for adaptation to tooth wear.

The tooth position is dynamic in continued eruption and also in the possibility of intrusion. If the mandibular closers are overactive, occlusion is no longer simply a stop for continuous eruption; it becomes a force which may cause the teeth to intrude.¹⁰⁰ Individuals with intruded teeth and consequent loss of vertical dimension are usually those who brace or clench the mandibular closing muscles. This is often related with psychological stress^{107, 109, 151} or stress of any kind. Frequently an individual in continuous pain will react by clenching the teeth. This continuous pressure on the teeth causes intrusion and loss of vertical dimension.

Decreased vertical dimension from loss of poste-

rior teeth is often corrected by partial dentures. The prosthesis may need to have its vertical height increased since there is frequently a "settling in" which may be due to a shift of the gingiva or to alveolar resorption. Resorption of the alveolar bone increases with aging. This is accentuated in long-term denture wearers and various types of bone disorders.

Increased Vertical Dimension

Increased vertical dimension interferes with the normal freeway space. The increased vertical dimension can be so great that the teeth are in occlusion when the mandible is in what should be the physiologic rest position. This stimulates the periodontal ligament proprioceptors and interferes with the criterion of the muscles being inactive in this position.^{66, 119}

An increased vertical dimension is nearly always due to dental restorations, fixed or removable prostheses, or bite planes that encroach on the freeway space. This may be caused by not evaluating the patient correctly for vertical dimension or, in some instances, by purposely increasing vertical dimension in an effort to "take strain off the temporomandibular joint." As noted above, applied kinesiology evaluation indicates that some patients who appear to require an increased vertical dimension in reality have an imbalanced muscular system or a neurologic tooth involvement which is sending false information about the true vertical dimension.

Vertical dimension for restorations or full dentures is usually determined by locating the rest position and then determining the freeway space which appears optimum for that patient. Another method of determining vertical dimension is by phonetics^{27, 121} (see page 372). This method measures the vertical distance during the production of speech sounds, usually the sibilants. The measurement of the closest speaking space prior to the removal of the natural dentition is reproduced in the dentures. There are various methods for determining the vertical dimension of speech when it is not possible to measure the original dentition.¹²¹

Whether the vertical dimension for prostheses is measured from the rest position or by the phonetic method, it is best to examine all the muscles of the stomatognathic system, as well as remote factors, prior to determining the vertical dimension. If there is an imbalance in the muscles of the stomatognathic system, the measured vertical dimension of rest or speech position will be altered. If these factors influencing the vertical dimension are corrected in the future by a physician's intervention or by the body's natural corrective processes, the prosthesis may no longer properly fit the patient. Failure to

Occlusion

correct pre-existing muscle imbalance and fitting the prosthesis to an abnormal condition may lock in the

abnormal function, causing health problems someplace else in the body.

IATROGENIC CAUSES OF MALOCCLUSION

There are many ways that therapeutic procedures can inadvertently cause malocclusion. Fortunately, many of these conditions are self-limiting because of the body's ability to be self-correcting and self-maintaining.

Manipulation

Manipulation of bony structure which requires contact of the skull can inadvertently create cranial faults. This may happen when the cranium is contacted to make an occipital correction, such as adjusting an occipital side-slip or occipital fixation. In some cervical manipulation techniques, the skull is contacted in a similar way. The problem seems most significant when contact is on the temporal bone in the area of the mastoid process. As was noted in Section I of this text, this is an excellent lever for making corrections in the cranial mechanism; therefore, it is also a lever which can impart improper forces into the cranium.

The mechanism by which this type of iatrogenic problem causes malocclusion can be from cranial faults changing the structural relationship of the cranium with the mandible, or secondarily by cranial nerves not properly controlling the muscular system. Changes in positions of the temporal bones and maxillae and their influence on the occlusion have already been discussed, as has been the muscular factor.

When using the skull as a contact point for occipital or cervical manipulation, a broad contact should be used to evenly disperse the force. Localized contact, such as directly over the mastoid process, places the force into the skull in a concentrated manner and may well be working a leverage factor into the closed kinematic chain of the cranium. After manipulation of the occiput or cervical spine, therapy localization and challenge should be used to evaluate whether any cranial faults are present or may have been created. If there are cranial faults, the usual approach as described in Section I of this text should be used to eliminate them.

It is easy to observe how improper cranial or cervical manipulation can adversely influence the occlusion. Without a thorough understanding of the interactions of the total body, it may be more difficult to recognize how improper manipulation in remote areas can cause similar types of malocclusion. If the

pelvis is manipulated improperly, it may cause failure of the normal interaction between the sacrum and cranium which must be present in the cranial-sacral primary respiratory mechanism; thus a cranial fault may develop from remote disturbance, which in turn causes malocclusion. Anything within the body that can influence the muscles of the closed kinematic chain of the stomatognathic system by way of proprioceptive control can likewise remotely cause malocclusion. This could be a foot, knee, or spinal subluxation; the list goes on and on. Often there is no recognition of the interaction between the cause and the effect. General manipulation of the remote area may well improve the symptoms in that area, but shift the stress to another location. The best way to avoid this type of iatrogenic problem is to always use the challenge mechanism, described in Volume I, to determine if and how any manipulation should be made. This method uses the body's own reaction to determine proper procedure; thus the physician is not attempting to impose upon the body that which appears to be a corrective effort but may not necessarily be appropriate under the circumstances and at the time of treatment.

Dental Procedures

Various dental procedures can also create cranial faults. Extraction of a tooth, whether from the upper or lower arch, can place forces into the cranium by way of the temporal bone or maxilla to create a fault. Similarly, forces used to seat a crown can also cause reactions in the skull. Mintz⁸⁰ discusses various dental techniques which can cause potential harm to the temporomandibular apparatus. These are basically any procedures imparting force into the TMJ. These same factors can also create cranial faults with the pressure introduced into the cranium through the mandibular fossae. The body will often correct these cranial faults with the balanced action of the mandibular elevator muscles seating the teeth of the solid mandibular arch into intercuspation with the flexible upper arch. Obviously it is the cranial faults not corrected by the body which become problems that create a malocclusion.

Restorations not accurately equilibrated can cause prematurities which put mechanical stresses into the cranium and stimulate the proprioceptive system to change muscle function. If the prematurity is singular or relatively simple, a new engram may be

rapidly built to change the synergism of the mandibular closers to bring the jaw into a clean centric occlusion, eliminating the prematurity. If complex, the nervous system cannot deal with the prematurities and confusion within the nervous system results. The muscles in the closed kinematic chain become imbalanced and the total complex is disturbed. Malocclusion can cause forces which create cranial faults to be placed into the skull with mastication and swallowing, thus complicating the total picture. These potential problems can be eliminated by evaluating the patient after restorations have been equilibrated, using applied kinesiology techniques described in Chapter 14. The cranial primary respiratory mechanism should also be evaluated for any disturbance which has developed. As noted later in this chapter, it is best to evaluate for cranial faults

and muscle imbalance and correct them prior to the design of prostheses and equilibration.

Prolonged dental procedures which require holding the mouth open may cause muscular imbalance.¹¹⁷ Usually the disturbance is to the neuromuscular spindle cell, which is easily corrected with applied kinesiology procedures. Even though these muscular involvements are often self-limiting, it is best not to take that chance; do the quick evaluation for muscle balance and make the necessary corrections. On some occasions, examination immediately after dental procedures may show a false negative because the anesthesia alters the normal nerve response. For this reason it is advisable to re-evaluate the patient for muscular imbalance and cranial faults on the succeeding office visit.

Considerations Prior To Equilibration

Equilibrating the teeth to proper centric occlusion — a delicate procedure — requires selective grinding and considerable skill. The loss of enamel is an irreversible process which cannot be taken lightly. Even though the usual processes require removal of only a very small amount of enamel, much unnecessary grinding can be eliminated by balancing the muscles in the stomatognathic system, using applied kinesiology techniques and perhaps occlusal bite planes, prior to equilibration.

The muscles of mastication may be the only ones requiring treatment, or a more in-depth correction may be necessary which includes the total stomatognathic system, or even areas remote in the body. Quite often it is necessary to make cranial corrections in order to balance the muscles. This is a two-fold benefit because it improves nerve function and also corrects cranial bone position, thus providing a stable, balanced base for the occlusion.

Failure to balance the muscles and provide a stable base for occlusion often means a patient must be re-equilibrated because of poor results or because the occlusion “keeps changing.” A more problematic situation occurs when a patient’s occlusion is equilibrated to an imbalanced skull containing cranial faults. This iatrogenically locks in the cranial faults, preventing the body from correcting them in its own self-correcting, self-maintaining way, or preventing a

physician from making the correction.

It appears that it is even possible to equilibrate an individual to an imbalanced skull when there are no cranial faults. This can happen when forces are applied to the skull during the equilibration process. Silverman¹²² developed a procedure to reduce this possibility during equilibration. He is particularly critical of forcing the condyle into the terminal hinge position, and of the head resting on the headrest of the dental chair. The headrest seems to be a particular problem if it is the older style V-headrest which tends to contact the mastoid processes of the temporal bones. The small amount of distortion which develops from these external pressures may not seem enough to cause a problem. It should be pointed out for those reading this text who are unfamiliar with dental equilibration procedures how small an amount of enamel is sometimes removed to eliminate the hit-and-slide of a prematurity. The changes that take place in equilibration from distortion of even a normal skull can, on a relative basis, cause considerable change in the occlusion and the apparent requirements in the equilibration process. Silverman’s procedures for eliminating skull distortion prior to equilibration will be discussed more thoroughly later, with a general outline of equilibration procedures.

SCREENING EXAMINATION FOR PREMATURITIES

This discussion is directed toward the non-dental physician to help determine when dental consultation is necessary. When an applied kinesiologist balances function in the stomatognathic system, some prematurities may be eliminated without further treatment. On the other hand, as changes are made in the stomatognathic system, sometimes prematurities develop as the result of cranial bone movement and change in muscle function.

Phonetic Occlusal Evaluation

When the engram of closing brings the teeth into centric occlusion without prematurity, there is a solid "click" as all the teeth intercusate simultaneously. There are several ways to observe the sound of teeth going into intercuspation.

The patient should be seated upright or standing, without his head against a headrest. These procedures evaluate the movement into intercuspation without any interference from the physician. The patient is asked to quickly elevate the mandible to "click" the teeth together. The physician first demon-



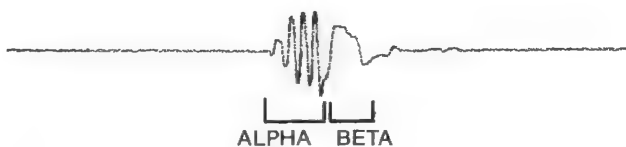
12—3. The best auscultation of TMJ dysfunction is with the bell piece of the stethoscope.

strates the activity to the patient by closing his jaw with a resounding clack. The patient repeats the action with the physician observing the sound, which can be done by simply placing an ear close to the jaw.¹³⁵ A better method is to place a stethoscope in the general area below the temporomandibular joint. There is less motion in this area to cause friction sounds on the stethoscope bell.¹¹³

The sound of teeth meeting in intercuspation can be recorded.¹²⁹ This can be done with a phonocardiogram or any recording instrument using a contact microphone as a transducer. Several variations of sound may be observed. High frequency signals represent a tooth or teeth hitting in prematurity with the cusps sliding over each other into intercuspation, called the alpha component. It is followed by a longer signal which is the beta component, representing the teeth coming solidly into intercuspation. An ideal occlusion consists entirely of a sharp single component as the teeth meet simultaneously.



12—4. Single sharp component of a normal occlusion.



12—5. The high frequency alpha component is the sound of deflective malocclusion.



12—6. The first sound group is popping of the TMJ. The second sound group is multiple contacts of deflective malocclusion.

In some cases there will be dual components as the teeth of one side of the jaw hit before those on the other side. When the two sides of the jaw hit independently, they may overlap each other and create some difficulty in interpreting the graph. Observing or recording the sound of occlusion on both sides helps to analyze the double sound. The louder alpha component will be heard on the side of prematurity. If the two sides of the arch are not hitting simultaneously, the side hitting first can usually be determined on a graph comparing the right and left sides.

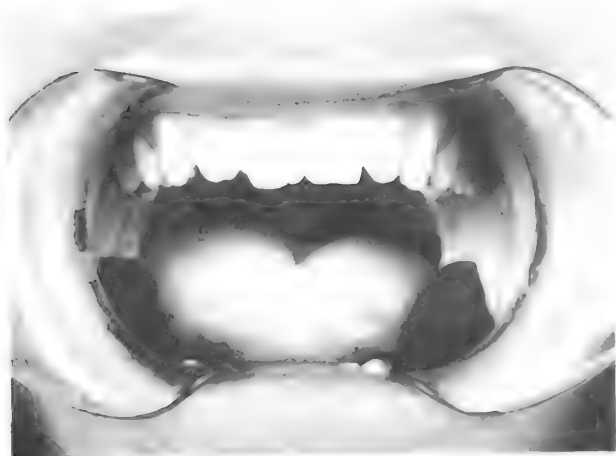
Palpation

Often the tooth or teeth hitting in prematurity can be localized by palpation.¹³⁵ The physician's finger is placed over the gingival tissues in the area where prematurity is expected. As the patient taps his teeth together, the teeth in premature contact move against the finger. There is usually excessive mobility in the direction of tooth displacement. The patient will be able to help guide this evaluation by indicating where he feels the first occlusion.

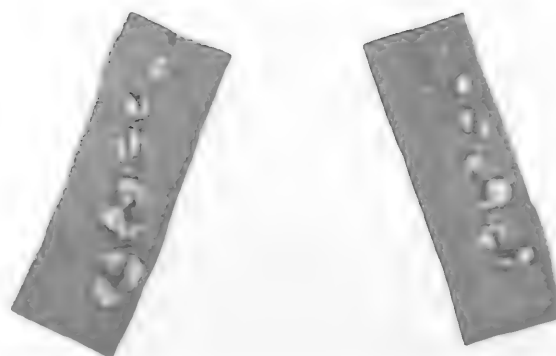
Palpation helps to evaluate prematurities in a dynamic manner. It must be recognized, however, that when the physician is palpating areas of the arches, the examination may considerably interfere with normal jaw movement. Information gained from palpation should be correlated with other tests for prematurity.

Wax Bite

A wax bite helps put in perspective the manner in which the teeth occlude by making an impression of



12—7. Green wax is placed over lower molars in preparation for evaluating occlusion. In actual practice, cheek spreaders are not used.



12—8. Impressions in occlusal indicator wax.

the occlusion in relatively soft wax. The wax is supplied in strips which are molded over the lower teeth. The patient moves into centric occlusion, making an impression in the wax. The wax is then removed and observed by transillumination as the physician studies the impressions against a light. In some cases it will be observed that the wax is almost totally perforated by some teeth and barely marked by others. This procedure is used by dentists to equilibrate new restorations. With the wax in place over the teeth, the premature areas can be marked with a pencil through the thinned area of the wax.

Marking Methods

There are several methods used to mark prematurities, the most common of which is the marking ribbon, supplied in different types. The ribbon is



12—9. Marking ribbon being placed between teeth to mark prematurities.

Occlusion

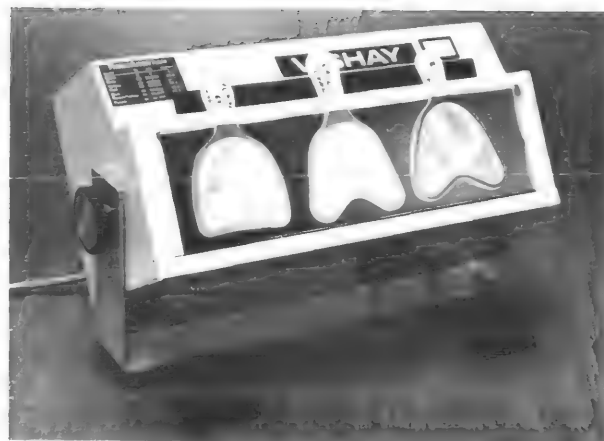
similar to carbon paper. It is supplied in different thicknesses and various colors which may be used for different purposes. The ribbon is held between the teeth and the patient taps his teeth together lightly, transferring the marking substance to the contacting teeth. The physician makes certain that the patient moves the mandible with the condyles in a physiologic centric relation position, guided by muscular balance. It is during this marking activity that some guide the mandible to the most superior retruded position for equilibrating the occlusion to the terminal hinge axis' centric relation.

Photocclusion

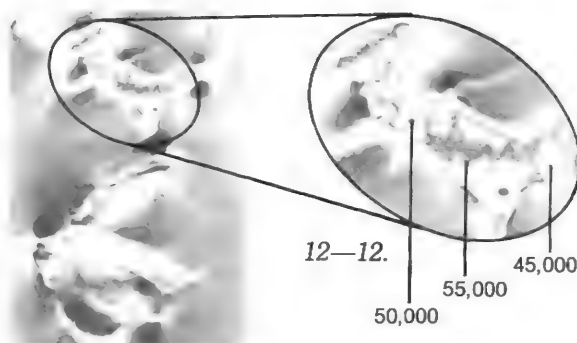
A recent development is the recording of occlusal forces on a plastic sheet which can be viewed through an optical instrument called a Photocclusion Analyzer™;* this determines the amount of compression between the various teeth. The force of the teeth in intercuspation distorts the plastic's molecular structure. The indentation caused in the plastic is then viewed through the optical instrument which causes different areas of the impression to produce different colors. This optical property of splitting the light beam into two other beams is called "birefringence."² A scale of the different colors gives the intensity level of contacts and percent of penetration, indicating the points of maximum contact. This provides an ability to quantify the forces applied in occlusion and to document the progress from therapy with a permanent record.^{1, 25}

The plastic is held in a frame; the complex is called a Memory Wafer.™ They are supplied in

*Photocclusion and Memory Wafer are the trademarks of Vishay Intertechnology, Inc., Malvern, PA 19355.

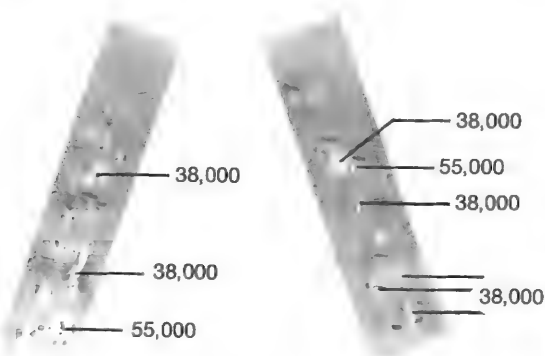


12-10. Photocclusion Analyzer.™

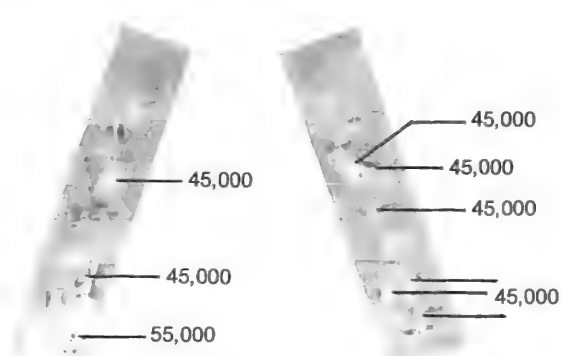


12-11.

Figure 12-11 is the impression of a molar. The enlarged section in 12-12 illustrates the shading of different amounts of force, which is determined by the color when viewed with the analyzer.



12-13. Before



12-14. After

Photocclusion of a patient under AK examination and treatment. The forces of occlusion, recorded here as pounds per square inch, balance in approximately one month as a result of balancing muscle function and tooth position remodeling. No equilibration was done.

different sizes to fit the patient's arches without interfering with soft tissue. It appears this method will be valuable in many types of research and in clinical practice.¹⁰⁴

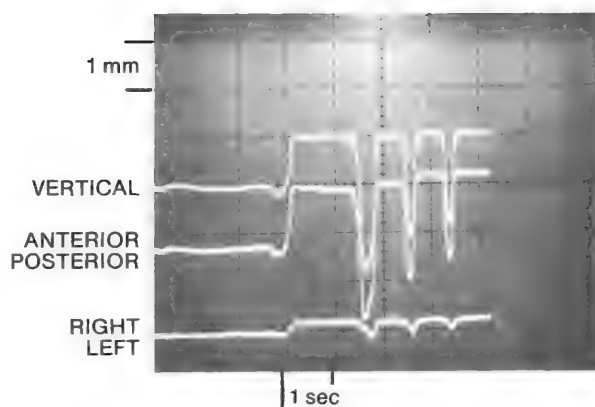
This unit has been used in our clinic to evaluate occlusion. It is often observed that a patient's major force of occlusion is limited to only a few teeth. These may be the back molars, anterior teeth, or anterior on one side and posterior on the other. Changes are observed in the occlusion from applied kinesiology treatment. This appears to occur as a result of change of muscle function, cranial bone position, and remodeling of the bone and periodontal tissues supporting the teeth. Information gained from this analysis has aided us in determining when referral to a dentist is necessary, and helps monitor the progress of the patient. Because of the recent development of the instrumentation, definitive reports cannot yet be made.

Mandibular Kinesiograph

The Mandibular Kinesiograph provides a dynamic means of determining how the teeth come into intercuspation when moving in the habitual closing arc as controlled by the engram of closing. (The Mandibular Kinesiograph is discussed on pages 319-325.) Treatment to the muscles and cranium, as indicated by applied kinesiology examination, can improve malocclusion, make it worse, or create malocclusion when none was present. If malocclusion is due only to muscle imbalance or cranial faults, it can often be eliminated immediately. This most often occurs from recent trauma. If there is good occlusion but muscle imbalance and cranial faults are present, their correction may create malocclusion; this can happen when a person has been equilibrated with problems present in the cranium or muscles. Graphs 12—15 through 12—20 show how the teeth may hit from a prematurity and slide laterally, anteriorly, or posteriorly.

Graph 12—15 demonstrates .2 mm of right slide into centric occlusion on initial tooth contact from the rest position. The slide is repeated when the patient taps the teeth together three times following the initial contact. Note also that the anterior slide becomes greater with each tap into intercuspation, yet the vertical dimension of intercuspation remains unchanged, indicating that the occlusion has a flat plane in which the teeth can slide anteriorly. This is sometimes created by equilibration and is called long centric.

The patient illustrated by graphs 12—16 through 12—20 sought the services of her dentist to relieve jaw pain and discomfort. Equilibration was successful in accomplishing that goal. At the same time she had



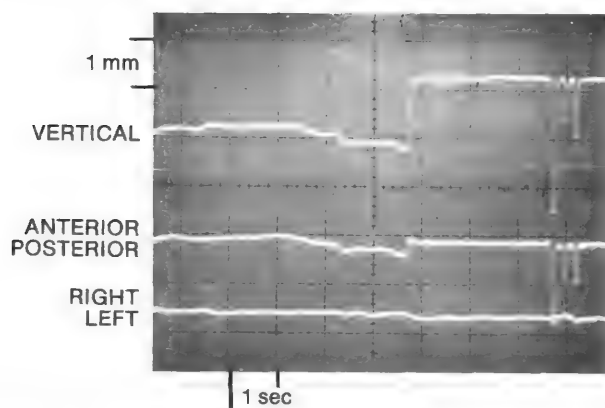
12—15. The top trace represents mandibular movement in the sagittal plane. The middle trace is anterior-posterior movement in the transverse plane, and the bottom trace is lateral movement in the transverse plane.

unrelenting upper thoracic spinal pain which was so severe she was unable to sleep. It was present to some degree all the time. Over several years she saw chiropractors, osteopaths, and medical doctors, with no success. Treatment consisted of adjustments, physiotherapy, vitamins, and medication. Two doctors diagnosed her condition as osteoarthritis of the spine. Applied kinesiology examination revealed severe imbalance of the trapezius and sternocleidomastoid muscles secondary to cranial faults. Cranial correction restored normal function to the muscles, as indicated by manual muscle testing. Unfortunately, the corrections were immediately lost when she forced her teeth into intercuspation and the muscles returned to the original imbalance. Apparently she had been equilibrated to the cranial faults; with the cranium corrected, malocclusion was present. Biting forced the cranium back to the distortion of the faults.

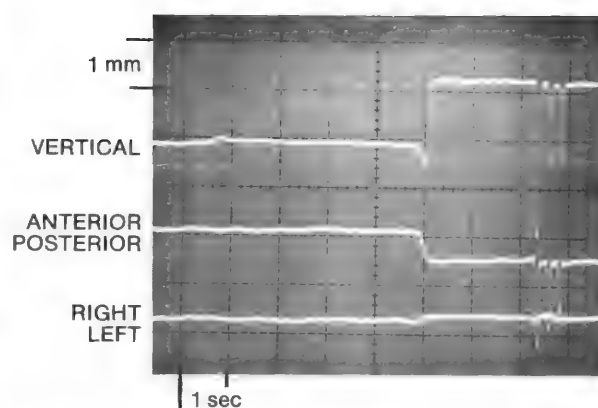
Unless malocclusion is very severe, it is good to attempt corrections without the use of bite planes or equilibration. Often the alveolar bone will remodel, allowing a slow adaptation of the tooth position to eliminate the malocclusion. If cranial fault correction does not hold, within four to six weeks bite plane therapy and equilibration will probably be necessary. Although the use of a bite plane speeds relief of symptoms, it usually prolongs the overall completion of the case. It is necessary to transition the patient out of the bite plane with equilibration and further adaptation. This was explained to the patient, and she agreed to work with the situation in the best way possible.

Cranial correction was made on a weekly sched-

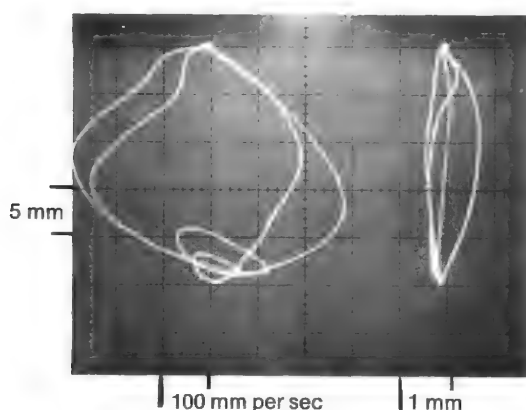
Occlusion



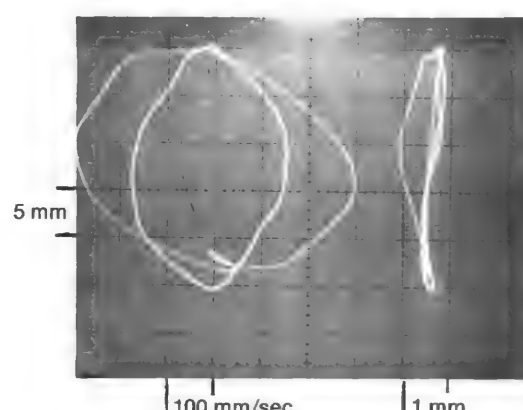
12—16. Prior to AK treatment. First half of graph is the rest position. Movement into centric occlusion is at the middle of the graph, and at the right is tapping the teeth together three times.



12—17. Immediately after AK treatment. Note the development of the posterior slide deflective malocclusion.



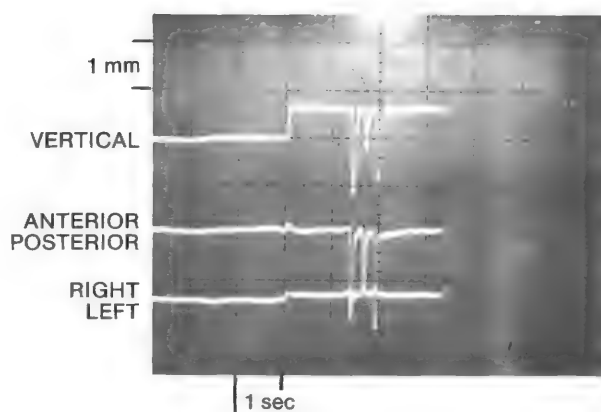
12—18. Prior to AK treatment. At left are two traces of vertical velocity. 1. Open-close. 2. Wide and fast as possible. Note the slowing by the engram as the teeth approach intercuspation. The right trace is movement in the sagittal plane.



12—19. Immediately after AK treatment. Increase in speed toward centric occlusion. There is a change in mandibular motion in the sagittal plane.

ule. There was no evidence of the corrections holding until the fourth week, which was also when she experienced the first relief from pain. When the cranium began to hold its correction, the jaw pain and discomfort which she had not had for several years returned because of the stress from malocclusion. The malocclusion was not severe; it continued to change, so the pain and discomfort were tolerable. Use of a bite plane or equilibration at this stage was not indicated.

Graphs 12—16 and 12—18 are prior to cranial correction. Some instability of the rest position in the vertical and anterior-posterior dimensions is shown in 12—16. There is minimal slide as the teeth intercuspate. Graph 12—17 is immediately after cranial correction. The rest position is stable, but now there is a posterior slide of .5 mm. Graph 12—20 is three



12—20. Three months after the initiation of AK treatment. The rest position is stable; movement into centric occlusion is good.

months later; the cranial corrections are stable, and there is no jaw or spinal pain or discomfort. The occlusion appears stable, with no prematurities.

Graphs 12—18 and 12—19 are velocity and frontal traces made before and after cranial correction at the same time graphs 12—16 and 12—17 were made. Note the increased speed with which the patient goes into intercuspation. There is also a shift from closing to the right before and to the left after the correction. This type of muscular function change is common immediately after applied kinesiology examination and treatment.

The combined use of the techniques described offers an excellent understanding of the occlusion, even for a non-dental physician. This is necessary to determine when dental consultation is required for completion of the case. None of the systems of evaluating prematurity are conclusive in themselves. The use of the wax bite or marking ribbon does not adequately evaluate the dynamic movement of the

teeth into intercuspation. The phonetic method gives additional information about how the teeth may hit and slide dynamically into intercuspation. Palpation of the teeth as they meet adds to this information. The Photocclusion method provides an ability to quantitate forces, but it gives little information about the dynamic movement into intercuspation. The Mandibular Kinesiograph gives this information, but it fails to locate the teeth of prematurity.

It is important to document prematurities in an initial examination. This does not mean that when they are found equilibration is automatically indicated. As will be seen in this discussion, the prematurities may change considerably as a result of muscle balancing, cranial correction, and other techniques used in applied kinesiology. In 1955, Perry⁸⁹ stated, "The evaluation of muscle function may some day be every bit as important as observing the intercusping of the opposing teeth in occlusion." We believe that day has come.

BITE PLANE-PLATE-SPLINT

There is confusion in the literature regarding the terminology for removable appliances which cover the teeth and are designed for various purposes. These appliances have been referred to as night-guards, biteguards, bite plane, bite plate, bite splint, guide planes, autorepositioning appliance, and many other terms. Here we will refer to them generically as bite planes unless a specific type is described; then we will provide a definition of it. These appliances are used for various corrective procedures, such as (1) eliminating the engram of mandibular closing, (2) interim balancing before more permanent occlusal change is made, (3) protecting the teeth from bruxism and supporting mobile teeth, (4) establishing vertical dimension, and (5) even improving the performance of athletes.¹²⁴

The bite plane may be on the mandibular or maxillary arch, or both. It is usually constructed of clear acrylic resin; sometimes wire is incorporated for added strength. This discussion of bite planes is limited to some of the more commonly used types. The discussion has a two-fold purpose: (1) to acquaint the physician not in the dental field with the purpose of bite planes and their fabrication, and (2) to put into perspective the observations made in applied kinesiology regarding bite planes, giving indications for use and, on a limited basis, the types most applicable when AK is used together with occlusal therapy.

Bite Planes for Neuromuscular Influence

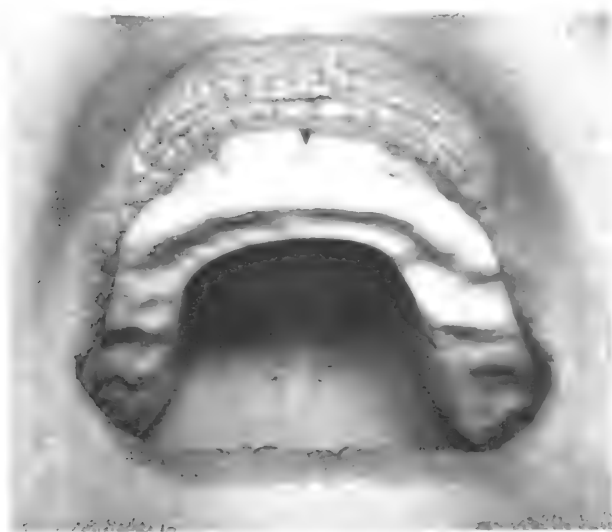
When the reason for bite plane therapy is to influence the neuromuscular system, it is done to eliminate pain or to return imbalanced masticatory muscles to normal. The purpose of the bite plane in this instance is to eliminate stimulation to the periodontal ligament receptors from deflective malocclusion. This is practical primarily when the malocclusion is such that there is conflicting sensory stimulation attempting to build an engram of closure to eliminate prematurities, which is impossible. For example, if the deflective malocclusion is such that information coming from the right side indicates the mandible should move to the left, and information coming from the left indicates the mandible should move to the right, the nervous system obviously cannot process the information to build a new engram of perfect mandibular closing into centric occlusion.⁹⁰

Many techniques and philosophies have developed for the fabrication of this type of bite plane. A common style⁵⁷ covers all the teeth in the upper arch, so there is no direct tooth contact on the incisal or occlusal surfaces. The acrylic resin is smooth on the occluding surface of the bite plane which meets the teeth of the lower arch. It is necessary for all the teeth of the lower arch to contact the bite plane so there is no intrusion on heavy contacts or super-eruption where there is no contact. The appliance

Occlusion

should have the basic principles of occlusion, such as disocclusion of the posterior teeth on lateral movements. If there are missing teeth, an acrylic block is incorporated into the bite plane to fill the space.

A bite plane of this type often provides almost immediate and dramatic relief of muscle spasm and pain. It is usually worn for one to two months prior to occlusal therapy. The patient is often requested to wear the bite plane day and night; philosophies differ as to whether the patient should wear it while eating. The length of time and consistency of wearing the bite plane are to reduce the effects of the engram of mandibular closing. This is accomplished by the bite plane eliminating tooth contact, which stimulates the periodontal ligament proprioceptors. The muscles now receive reduced influence from this mechanism, and the engram control of mandibular movement continually lessens with time. With the reduction of conflicting afferent stimulation from the malocclusion, the muscular hypertonicity lessens. If this is the only factor causing the muscular imbalance, the procedure is dramatically effective. If there are additional factors the procedure will only be partially effective, or it will provide no change in the muscular spasm.



12—21. Maxillary bite plane.

Here is a good opportunity to illustrate a primary and a secondary condition and the response to therapy, depending on which is being treated. When there is improper stimulation to the periodontal proprioceptor, such as in applied kinesiology's neurologic tooth condition, or if malocclusion is complex and cannot be dealt with, imbalance will develop in the masticatory muscles. Prolonged muscular spasm of this type can create dysfunction in the neuromuscular spindle cell, which in turn

causes further muscular imbalance. Under these circumstances, there are two conditions present. Stimulation to the periodontal proprioceptor is primary, whether from malocclusion or dysfunction of the proprioceptor itself. The secondary condition is the dysfunction of the neuromuscular spindle cell in the muscle, which may have become severely hypertonic as a result of the primary periodontal proprioceptor disturbance. Treatment to the muscle spindle cell will either give only temporary relief or be totally ineffective. The proper therapeutic approach is to relieve the stimulation to the periodontal proprioceptor. In the case of malocclusion, this can often be effectively done with bite plane therapy.⁶⁵ When the periodontal proprioceptor itself is at fault, bite plane therapy may be successful in some cases and not in others. The best approach is to manipulate the tooth with the technique discussed on page 266.

Reversing the example, trauma to a neuromuscular spindle cell in a muscle of mastication can be primary, with the resulting hypertonic muscle causing malocclusion. Now stimulation to the periodontal ligament is secondary. In this case, bite plane therapy will probably be ineffective in relieving the muscular spasm. The appropriate approach is to locate and treat the dysfunctioning neuromuscular spindle cell, correcting the primary problem. The only way that bite plane therapy can be successful here is if eliminating stimulation to the periodontal proprioceptor takes enough stress off the muscular imbalance for the body to correct the neuromuscular spindle cell with its own recuperative ability. Many of these conditions develop into a vicious circle. Breaking the circle is often an effective method of treatment, even though the therapy is not directed to the primary problem. In this example, the body may very well be able to correct the traumatized neuromuscular spindle cell without additional factors present. When the muscular imbalance becomes even greater because of the change of engram as a result of complex malocclusion, the muscles may become more hypertonic, perhaps to the state of spasm where the body can no longer correct the traumatized neuromuscular spindle cell.

In a vicious circle such as described above, treatment is often directed to secondary effects rather than to the primary cause. The patient often chooses the type of treatment which will be provided when he chooses a physician. Often the primary factor in diagnosis is that you do not recognize what you do not know. Even though symptoms can often be improved or corrected by treating effects, the best, most rapid and complete correction is obtained when the primary involvement is found and corrected. Again, it is obvious that differential diagnosis is

called for when there is imbalance of the stomatognathic system. There is no single therapeutic approach which can be considered a panacea for what is often called the temporomandibular joint syndrome. As Farrar³⁵ states, "Whoever has ever heard of Hip syndrome? or the Knee syndrome?" *We would not think in terms of treating a painful knee with only one therapeutic approach, but in many offices the so-called TMJ syndrome consistently receives the same therapeutic approach for every patient.*

Clinical evidence indicates that individuals deriving almost immediate and dramatic relief from pain with a bite plane are those who have improper stimulation to the periodontal proprioceptors which is primary and causes conflict within the neuronal pools, resulting in muscle spasm and pain. If the problem has created a secondary disturbance in the muscle's proprioceptors, it takes a longer time for the bite plane to be effective, if it is going to be so.

With a bite plane, the mandible no longer maintains a specific position during occlusion; there is freedom for it to move with the muscular change that develops as a result of the engram breakdown. Kovalski and DeBoever⁶⁹ demonstrated that the mandible typically moves anteriorly and laterally with one month's use of an occlusal bite plane that has sufficient centric freedom built into the appliance. This reveals how the engram controls muscles to bring the teeth into centric occlusion. With the abolition of proprioceptive stimulation in the periodontal ligament, the muscles relax to allow the mandible to move into a position of physiologic centric relation in harmony with relaxed muscles.

The previous description of the use of bite planes tells of their supplemental use in preparing for the ultimate corrective dental therapy. They are used to break down the engram and reduce muscular spasm prior to equilibration of the occlusion. This effort recognizes that muscular imbalance may be predominant in the malocclusion. It is important to remove the muscle spasm, whether the equilibration is being done to match centric occlusion to centric relation with the condyle in the most retruded superior position of the fossa, or to match physiologic centric relation. Even though equilibration in the retruded superior position is not recommended in this text, it seems important to point out that muscle spasm complicates bringing the condyle into this CR position. Equilibration to physiologic centric relation can **only** be done in the presence of balanced normal muscle function, since that is what determines the position.

Bite Plane to Supplement Therapy

Another use of the bite plane as an interim

therapy is when there is complex dysfunction within the stomatognathic system and throughout the total body. In this case it may be necessary to temporarily take the effects of malocclusion out of the picture so that treatment can be administered to other areas over a long enough period to gain stabilization of the corrections. After all other areas of the stomatognathic system and body have been corrected and stabilized, the occlusion is equilibrated to this normal activity. Failure to reduce the disturbing effects of malocclusion can prevent other corrections from holding by neurological or mechanical means.

The neurological problem occurs because correction of cranial faults and other areas in the stomatognathic system and body can cause many changes in occlusion over a few weeks to which the body cannot adapt. This rapid change of occlusion may present many types of malocclusion which would continually stimulate the periodontal proprioceptors, causing a continuous body effort to regulate the muscles of mastication in varying ways. It may be impossible for the body to adapt to this rapid occlusal change. A bite plane eliminates intercuspation and can be temporarily used to take the occlusion out of the picture while rapid changes are taking place.

Under normal circumstances the occlusion is continuously changing, but only very slightly. The engram is regulated to match this change by the afferent information, bringing the muscles into an optimum balance for the occlusal change taking place. Force directed to the occlusion during mastication and swallowing continues to reinforce this engram. Under normal circumstances the occlusion changes by remodeling of the alveolar arches and temporomandibular joint to provide as optimum an occlusion as the structure will allow.^{9, 10, 81} Since the natural process of adapting the occlusion to the current status of total body function is slow, it is sometimes necessary to exclude the occlusion from total interaction while other corrections are obtained. The malocclusion may later be equilibrated by a dentist to match the corrected structure of the rest of the body.

The second reason to use a bite plane as supplemental therapy relates specifically to the cranial primary respiratory mechanism. Although we have differentiated two purposes — neurological and mechanical — for a bite plane during interim therapy, both purposes are usually involved. The mechanical aspect requires some additional consideration regarding the purpose of the bite plane. Whereas the neurological factor is only to eliminate periodontal proprioceptor stimulation, the mechanical aspect requires a balancing of the jaws to prevent transmission of imbalanced forces into the cranium.

Occlusion

A short review of the effects of mastication on the cranium helps in understanding the purpose of the bite plane in mechanical considerations. The muscles of mastication pull with a great amount of leverage into the cranium. When the cranium is mobilized by this force, it can either be detrimental or advantageous to the cranial primary respiratory mechanism. This can often be observed in an individual with cranial faults by simply having the person stretch his jaw wide open several times. After this activity evidence of the cranial faults will often be removed, as observed by both therapy localization and challenge. This "correction" is usually just temporary, especially when malocclusion is present. It does show the influence of the skull muscles on cranial function, but the temporary correction will most often be immediately lost as soon as the individual bites down or swallows.

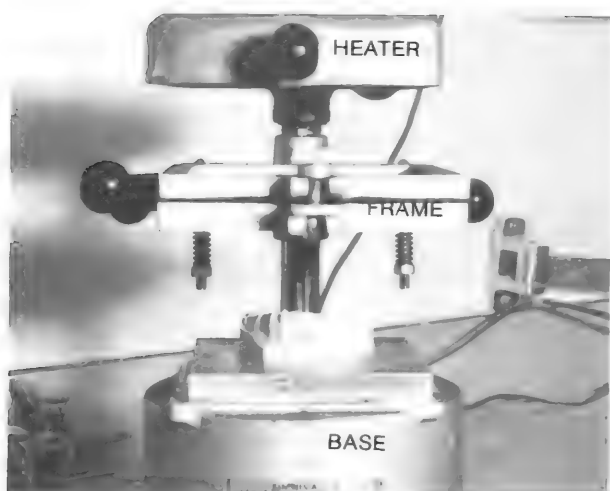
Another example of the influence of occlusion and the muscles of mastication on the cranium is the ability of swallowing and chewing to immediately eliminate cranial imbalance.¹²² This occurs in an individual with normal occlusion and balance of the masticatory muscles because the mandible is a relatively solid base to which the movable maxillae are fitted and pulled into position by the muscles of mastication. This may be a difficult concept to grasp for those who have been working with occlusion. Generally, the skull is considered the fixed base and the mandible the movable structure. The reason the mandible must be considered the fixed base is that the lower dental arch is in a single bone, making it more solid, whereas the upper dental arch has the teeth of one quadrant in the right maxilla and the teeth of the other quadrant in the left maxilla. With the understanding that the skull is movable and requires this motion for function, the upper dental arch becomes the physiologically movable structure and the mandible becomes the more solid base; thus the teeth in the two halves of the upper dental arch can move to meet the lower dental arch for complete intercuspation as the mandible is forced into closing.

The action of the muscles in this activity must be taken into consideration. What is usually the origin of the muscle changes to the insertion, with the mandible being the stable base of origin. The insertion of the muscles pulls on very important leverage factors. The levers into the sphenoid, a key bone, are the pterygoid processes which are pulled on by pterygoid muscles. The zygomatic process is the lever into the temporal bone, pulled on by the masseter muscle. The temporal bone is also influenced by the temporalis pulling on the potentially flared squamous portion of the bone. This is a marvelous corrective mechanism of the body for its

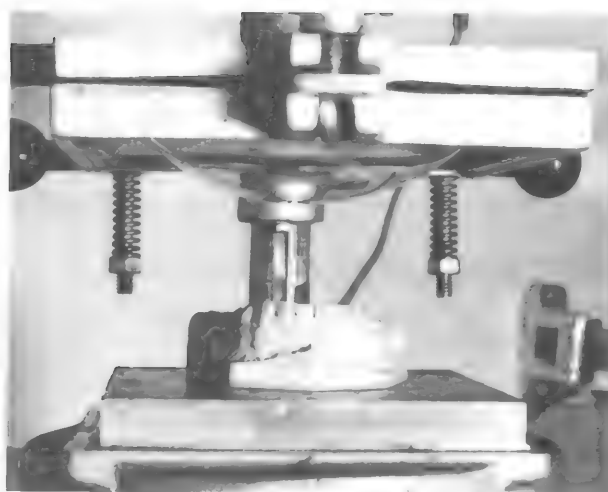
own self-protection and self-correction. Introduce malocclusion or muscle imbalance, and it becomes obvious that the same mechanisms which are ordinarily corrective can become detrimental to the cranial primary respiratory mechanism.

An example is cranial faults which have developed over a prolonged period. The occlusion may be functional because remodeling has positioned the teeth to fit the imbalanced skull. Correction of cranial faults may change the relation of the maxillae and temporal bones in such a manner that the mandible no longer moves into a position of centric occlusion without deflective malocclusion. The change may be relatively simple, with the mandible shifted only slightly to one side because of the relative change in the two fossae or different activity of cranial nerve V. In this case a new engram can rapidly be built by the body with minimal disturbance to normal function. On the other hand, the maxillae may change their relation, causing a wider or narrower upper arch, producing deflective malocclusion on both the right and left sides in opposite directions. When this happens, the teeth of the more rigid mandible force the maxillae into the position prior to cranial correction as they intercuspate with the upper arch. Under these circumstances the cranial corrections are lost almost as rapidly as they are accomplished. Often it is not even necessary for an individual to bite down hard. The intercuspation present in swallowing may return the cranial fault. When a cranial fault returns after biting down or swallowing, it is not always due to this mechanism. (The differential diagnosis of why the cranial fault returns is presented in Chapter 16.)

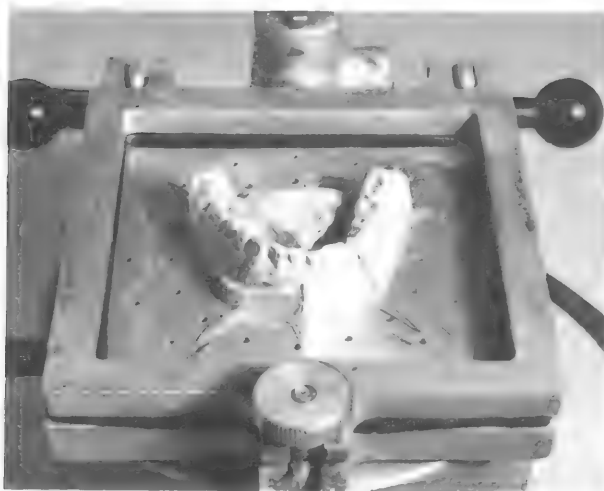
A bite plane can provide the method for preventing the return of a cranial fault under these circumstances. Its construction and use are modified from that described previously. First it is necessary that the bite plane be fabricated for the mandibular arch. This varies from the most common procedure, which is the maxillary bite plane, used because it is a stronger appliance and sometimes creates less interference with the anterior teeth. The primary advantage of the mandibular bite plane is that it avoids interference with normal cranial motion. The maxillary bite plane often fits over the teeth with great accuracy. This locks the maxillae together so that they cannot function independently, as they must do in the cranial primary respiratory mechanism. Patients are sometimes referred to an applied kinesiologist when a maxillary bite plane has already been fabricated. It is often observed that cranial faults return immediately after the maxillary bite plane is put back into the mouth, even without biting. This writer has seen patients totally relieved of a



12—22. Omnivac Vacuum Adapter. In the base is a vacuum pump. The frame holds a sheet of resin while the heater softens it.



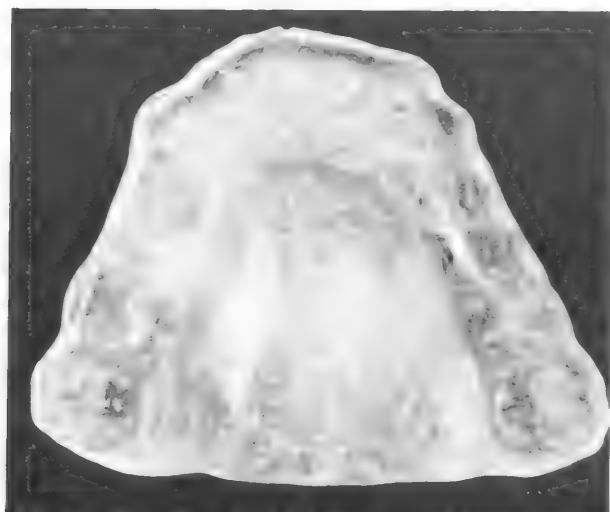
12—23. Note the heated resin drooping from the frame which is about to be lowered to the patient's model.



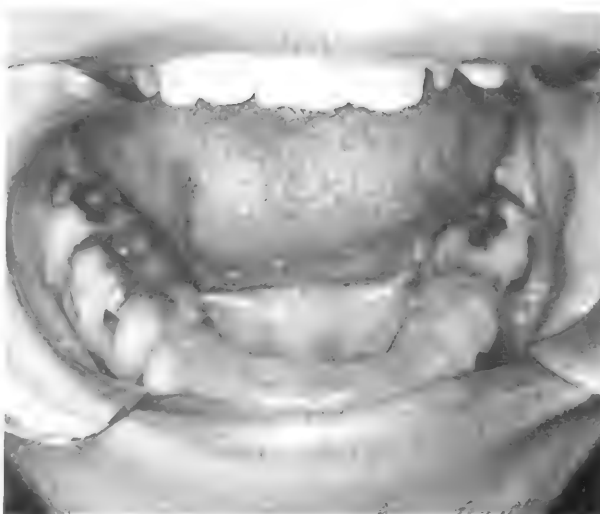
12—24. The resin is vacuum-formed over the model.



12—25. Cutting away excess resin.



12—26. Unfinished mandibular bite plane.



12—27. Mandibular bite plane.

Occlusion

severe headache by cranial correction, only to have it return within thirty seconds of re-inserting a maxillary bite plane.

The second consideration in the mechanical use of a bite plane is that it sometimes needs to be re-equilibrated as the cranium and occlusion change. The same basic principle stated previously regarding the necessity of all teeth occluding to the bite plane when in physiologic centric relation is applicable here. Sometimes cranial corrections and balancing of the muscles of the stomatognathic system and body cause some teeth to contact the bite plane prior to others. In this case, the same cranial fault re-creation problem exists as was present without the bite plane. Ideally, the dentist who installed the appliance is familiar and works with the cranial primary respiratory mechanism and can adjust the bite plane immediately after making cranial corrections. Sometimes it is necessary for two physicians to work together, one correcting the cranial mechanism and the other adjusting the bite plane. Usually this is complicated by the doctors being in different offices. In this case, if the appliance needs adjusting to prevent the return of cranial faults, the doctor who has corrected the faults should place cotton rolls between the molars on both sides so that the patient cannot come into occlusion while traveling to the dentist's office for equilibration of the appliance. This prevents the cranial fault(s) from recurring prior to the equilibration.

Whether the bite plane is used to relieve stimulation of the periodontal proprioceptor or for the mechanical reason, or both, the follow-up procedure is basically the same. The bite plane is used twenty-four hours a day for as long as it is necessary to obtain a lasting balanced correction of the cranium, muscles of the stomatognathic system, and any remote problems which may be influencing the area. This usually takes four to six weeks, but it may take a greater or lesser amount of time. Care should be taken that the corrections have actually been accomplished, because the next step is equilibration; this is a permanent process requiring selective grinding, with the resultant loss of enamel. It may also be necessary to provide for missing teeth or reconstruction of the present dentition.

Establishing Vertical Dimension by Use of Bite Planes

The literature reveals considerable controversy about vertical dimension. Some take the position that almost everyone requires a vertical dimension increase because the general population has lost mandibular size from genetics and malnutrition; there are also those who state that no one needs an

increase in vertical dimension. As with so many other controversies about the stomatognathic system, it seems that those who take a positive stance regarding nearly all patients have such an extreme view that it is untenable. Somewhere between these two extremes is the most appropriate approach. A thorough understanding of the interactions within the total body helps put this into perspective.¹²³ Again, diagnosis of the condition is the necessary determinant.

Studies done over the years representing both pro and con with regard to "bite opening" help put in perspective a moderate view on this controversial subject. It was shortly after Costen's²² indictment of loss of vertical dimension as the cause of deafness and sinus symptoms that bite opening became very popular. Although Costen's hypothesis for the symptomatic picture has been discredited,^{110, 112, 116} there does appear to be a correlation between these symptoms and disturbance in the stomatognathic system. Current knowledge indicates that dysfunction in the auditory system and sinuses can develop because of cranial faults, as described in Section I; there appear to be other forms of neurologic disturbance which cause auditory problems.^{3, 4, 56} Costen's primary hypothesis was related to the anatomy of the temporomandibular joint. Dysfunction was supposed to encroach upon the eustachian tube, with the condyle exerting pressure on or near the auriculotemporal nerve and in some cases causing an erosion of the tympanic plate, influencing the chorda tympani nerve, among other factors. Evidence of temporomandibular joint dysfunction affecting hearing is observed by improvement of the audiogram and tympanogram in applicable cases.^{3, 4, 48}

Although these disturbances are no longer necessarily related to a decreased vertical dimension, there is some continued controversy about the joint's anatomy. There are those who describe the joint as not being designed for weight bearing^{84, 100, 105} and those who adamantly state that it is a strong joint designed for weight bearing.^{24, 118} Barbenel,⁸ from an analysis of the force actions of the temporomandibular joint, concludes that the joint is load-bearing during function. Sicher's *Oral Anatomy*³¹ considers that the fossa is not a stress-bearing part of the temporomandibular joint, but then concludes that the lack of blood vessels in the firm central portion of the articular disc clearly demonstrates there is considerable pressure in the joint. It seems that a combination of the two philosophies of the joint is proper for its unique type of function. The sliding, non-lever-type motion of the TMJ described on page 326 requires a weight-bearing factor of **the disc and articular eminence** during translation. This, com-

bined with the non-lever action of the joint,^{44, 105} indicates no great need for a weight-bearing factor in the fossae during the strong forces of mastication and the continual action of swallowing throughout the day and night. The teeth and the articular eminence take the massive amount of accumulated forces. This does not take into consideration loss of posterior support from the teeth. This support is necessary for the non-lever action of the stomatognathic system to function normally. The teeth and the muscular non-lever action of the mandible protect the temporomandibular joint. This can be observed on a dry skull by simply placing the mandibular arch into centric occlusion with the maxillary arch. The distance between the condyle and mandibular fossa is filled by the disc and soft tissue. The only way that excessive force can be placed into the temporomandibular joint is by disocclusion of the teeth. If there is loss of posterior support, the condyle can be jammed into the fossa. **Now** the argument which states the TMJ is not designed for weight bearing becomes appropriate. **No longer** is the joint protected by the non-lever function of the maxillary and mandibular arches working together.

Temporomandibular joint disturbance does not always indicate that there is a "jamming" at the joint. Gelb and Arnold⁴⁰ point out the importance of diagnosis prior to determining the therapeutic approach by stating, "Unquestionably no single factor can be responsible for all (the) varied symptoms. It is the (interaction) of the nervous, muscular, and vascular systems, at least in the affected region, which requires our attention."

History shows the adverse consequences which can develop as a result of indiscriminately increasing vertical dimension or, as it is sometimes called, "opening the bite." At one time it appeared to be a significant enough problem that there was an editorial in the *Journal of the American Dental Association*³² which cautioned the profession that opening the bite threatened to assume the proportions of a fad. Many articles appeared, usually with case presentations of the problems which developed. Most pointed out that the muscles were dominant in the function,⁷⁸ and that special training was necessary in determining vertical dimension changes.¹¹¹

When the vertical dimension is increased inappropriately, it causes clenching and grinding of the teeth. The forces appear to be more severe at night because the restraining forces of gravity and conscious control are absent.¹¹⁹

With long-standing use of bite planes or other appliances that fill the freeway space, there may be intrusion of the teeth¹⁴⁴ because the balance between

occlusal and eruptive forces is changed.

The importance of maintaining normal freeway space cannot be overemphasized. The muscles function within a range, and if freeway space is excessively increased or decreased, muscle dysfunction develops with the attendant damage to the dentition. It is not only encroachment upon the freeway space that is of concern; increased freeway space also causes muscular disturbance. Jarabak⁶⁶ demonstrated that both an increase and decrease of freeway space caused muscle hyperactivity on electromyography. The study was done by evaluating individuals with dentures made to three different vertical dimensions. Overclosure caused spontaneous hyperactivity, and excessive vertical dimension caused increased muscle tension.

Bite planes used for eliminating stimulation to the periodontal proprioceptors and for mechanical balancing of the skull to compensate for cranial corrections, as well as those for stabilizing the teeth which are discussed later, change the vertical dimension by covering the teeth. Care should be taken that this does not create a new problem of muscle spasm, confusing the diagnostic process. Weinberg¹⁴⁵ discusses this cause of muscle spasm and chronic muscle fatigue, relating to the fact that paradoxically the bite plane, although an iatrogenic sustaining mechanism, often gave some symptomatic relief on insertion.

The optimum approach in considering vertical dimension is that of bite restoring, not bite opening. Several factors must be taken into consideration in determining if **bite restoring** is necessary and applicable at this time. When there is loss of posterior support from loss of teeth, the answer becomes obvious. As previously noted, the muscular levering action is changed simply from no teeth to act as the fulcrum point.

Loss of vertical dimension may appear to be present as a result of excessive wear on the posterior teeth. Most often there is no loss of vertical dimension in these patients because the natural eruptive process of the teeth maintains the dimension. The force of eruption is from additional layers of cementum on the root and vertical development of the alveolar process.²⁴ As cementum degenerates with age, it is covered by a new layer of young cementum. This adding on process requires space which is provided by the continued active eruption of the teeth, which depends on continued occlusal and incisal wear; thus wear is essential for the dentition's health.³¹ Teeth will continue to erupt until there is a stop to prevent further migration.

The balance of the muscles provides the stop for tooth eruption. Unless there is muscular hyper-

Occlusion

tonicity, the worn tooth will continue to erupt, maintaining vertical dimension. In the presence of normal muscles, freeway space and vertical dimension are regulated by muscle activity. The stop for eruption is created more by tooth contact from frequent swallowing throughout the day and night than from mastication.

Loss of vertical dimension can be from intrusion of the teeth.^{100, 144} This is caused by continual clenching which provides more force than the force of eruption can compete with. Just as teeth are moved by continual pressure from orthodontic appliances, the teeth intrude to cause a loss of vertical dimension. In these patients there is usually a high stress situation causing the continual clenching. When this occurs, increasing the vertical dimension by bite plane therapy, partial dentures, or restorations is contraindicated. The muscular bracing should first be eliminated, or there will just be further intrusion of the teeth.

Correct vertical dimension is an individual consideration. It is influenced by the balance in the closed kinematic chain of the stomatognathic system. Rules indicating there should be so many millimeters of rest position have been described as dangerous⁷⁸ because the bony structure and optimum length at which the muscles function are individual; they do not change to meet predetermined criteria being imposed upon them by the physician.

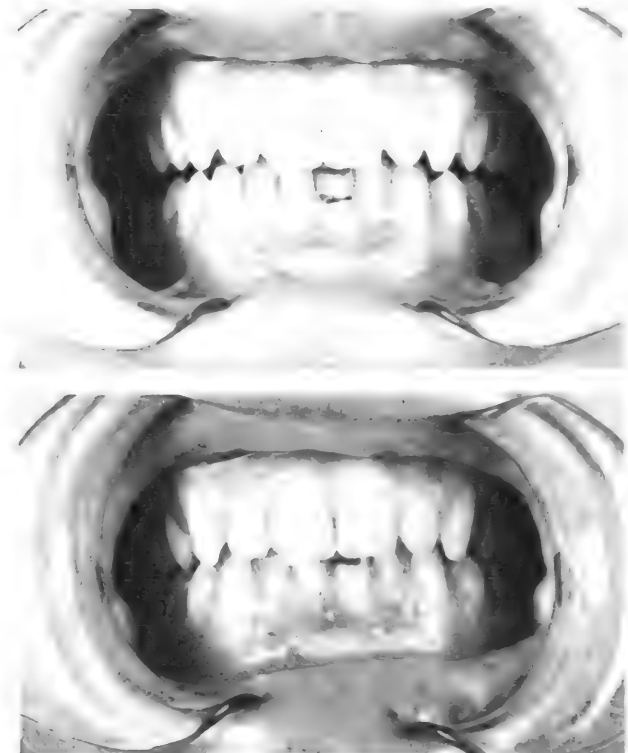
There are two reasons for determining the vertical dimension. One is for the planning of prostheses, such as full or partial dentures, or for restorations. In this situation, there is evidence that the present vertical dimension is correct, and the effort is directed toward reproducing it in the design of the prosthesis. The second situation is where the vertical dimension is not correct as a result of loss of posterior support.

Many methods have been described to determine optimum vertical dimension. One of the earliest procedures was to determine the rest position and then add a specific measurement for freeway space to arrive at where centric occlusion should be.⁸⁶ The first problem in this procedure is locating the exact rest position. Techniques vary from simply asking the patient to assume a resting mandibular position and then measuring the freeway space to the more sophisticated method of using modern electrophysiologic techniques. Electromyography can be used to find the position where the muscles are inactive, and the freeway space is then measured.^{90, 119} The Jankelson⁶³ Myo-monitor⁸⁵ is used to relax the muscles to their apparent normal resting length for measurement of freeway space.⁴³ The Mandibular Kinesiograph⁸⁵ is used to obtain accurate measure-

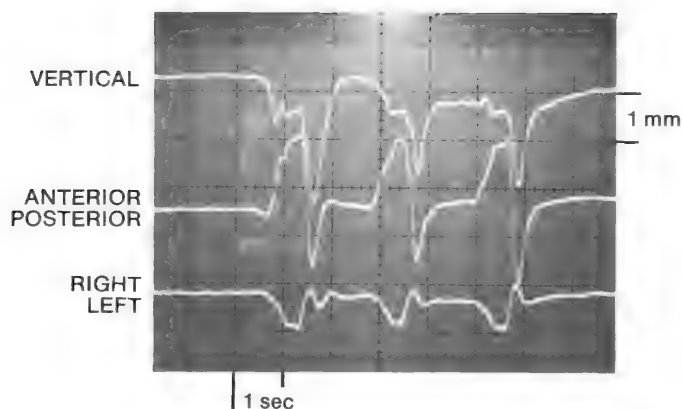
ment of the mandible's position; it can be used in conjunction with electromyography and the Myo-monitor to determine rest position.

Silverman^{120, 121} described a method using phonetics to reproduce the vertical dimension. The freeway space method requires complete relaxation of the patient's muscles to find the rest position. In the phonetic method, the patient's mandible is brought into action and a dynamic measurement is made. This method is easily accomplished and has excellent reproducibility at different times and by different operators. The effect of applied kinesiology treatment on the stomatognathic system can be observed by the changes in the vertical dimension as observed by the phonetic method. Because of this potential change the individual should be examined and any corrections made prior to measurement.

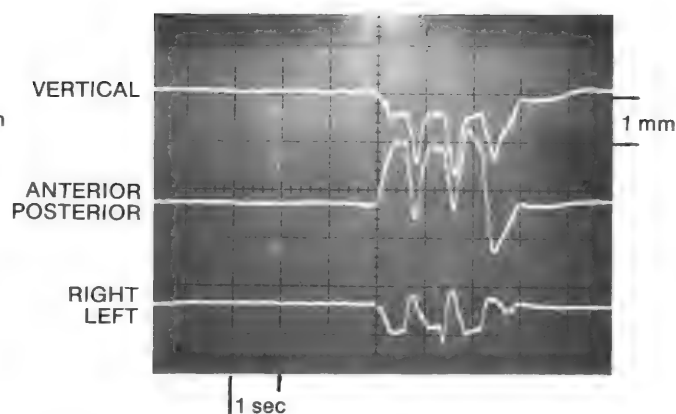
The steps for recording vertical dimension, with slight modifications from Silverman's original description, are as follows. (1) The patient is in an upright seated position without his head on a headrest. If the muscles of the total stomatognathic system are balanced, the head will be in a neutral position with the occlusal plane horizontal. With the patient in centric occlusion, a line is marked on the lower central incisor at the level of the incisal edge of the



12—28. Top illustration represents the closest speaking line. The lower line shown in the lower illustration marks intercuspsation, which is the centric occlusion line. Distance between the lines is the closest speaking space.



12—29. Repetition of the word “Mississippi” prior to AK treatment. Note first vocalization ends at centric occlusion. There is generally poor reproducibility of mandibular movement.



12—30. Immediately after AK treatment. The reproducibility has improved considerably.

upper central incisor. This line is called the centric occlusion line. (2) The patient is requested to say the word “yesss” with emphasis on the “sssss.” The position of the mandible when this sibilant is pronounced is called the “closest speaking space.” A second line is drawn on the lower central incisor at the level of the upper incisal edge. This is called the “closest speaking line.” The distance between the centric occlusion line and the closest speaking line represents the closest speaking space. (3) The patient is now asked to read some material rapidly, quickly say the word “Mississippi,” or repeat “66.” With this more rapid action, the examiner watches the incisal edge of the upper central incisor to determine if it repeatedly comes to the closest speaking line every time the “s” is pronounced. This quickness of action places the mandible in involuntary control, which should be continually repeatable to the line. It may be necessary to slightly adjust the closest speaking line until repetition is present. It is important that the words “Mississippi,” “66,” or reading be done at the same speaking level, as a change in volume reduces the reproducibility.²⁷

The closest speaking space is reproduced in the denture or restoration. Additional procedures are described by Silverman when natural dentition is not present prior to recording vertical dimension.

In our laboratory the Mandibular Kinesiograph⁸⁵ is used to measure the closest speaking space. Even though this is considered a reproducible measurement, the effects of applied kinesiology treatment to balance the stomatognathic system are shown in figures 12—29 and 12—30. The interaction of the stomatognathic system is clearly demonstrated here, since the treatment that effected the change was not directed to the muscles of mastication.

When there is a need to restore lost vertical dimension, a greater problem is present than just described, where vertical dimension is correct and is only to be recorded for duplication. Determining rest position and the closest speaking space, along with applied kinesiology procedures, can help to ensure that the modified vertical dimension is correct.

Of primary importance in modifying vertical dimension is that normal freeway space not be encroached upon and that no teeth touch at the closest speaking space. If the normal freeway space is reduced, muscle spasm will result.³¹ Not only will there be harm to the teeth;^{24, 78, 111, 119} even more important in the context of this text, there will be disturbance to the balance of the closed kinematic chain of the stomatognathic system with a resultant wide range of effects. Ironically, sometimes increasing vertical dimension will at first give considerable relief to the patient, even though it may be increased too much. This is apparently due to the increase compensating for some other factor to temporarily bring balance into the system. We can compare this with the relief a patient sometimes obtains as a result of putting a heel lift under an apparently short leg when the leg is, in reality, short due to a pelvic misalignment. The heel lift gives compensation to the problem, but it does not correct the basic underlying cause. Correction of the pelvis is obviously getting to the primary cause of the problem rather than treating the secondary effects.

The bite plane used to increase vertical dimension can be called an occlusal level adjusting bite plane. This appliance is a temporary measure used on a trial basis to test the revised vertical dimension for compatibility with the closed kinematic chain and

Occlusion

temporomandibular joint.^{41, 70} It is used on a continuing basis as other aspects of the stomatognathic system are corrected. It may need adjustment as cranial faults are corrected and balance is developed in the closed kinematic chain. Throughout the use of the appliance, the patient is evaluated for adequate freeway space and for tooth clearance at the closest speaking space. Examination at the beginning of treatment may indicate the patient needs an increased vertical dimension, but as various factors are corrected in the closed kinematic chain and remote areas of the body, the indication for additional vertical dimension is diminished; finally, no need of increase may be indicated as other corrections are obtained. Additional applied kinesiology methods of testing vertical dimension are covered with the AK examination and correction in Chapter 14.

Bite Planes for Protection of the Teeth

Bite planes are sometimes used to protect the teeth in periodontal disease and from bruxism. Avoiding tooth movement in periodontal problems is the primary purpose of the bite plane, which is usually called a splint in these cases. The only consideration necessary in this discussion is whether the stomatognathic system may be thrown out of balance by a change of vertical dimension with the addition of the splint. To adequately splint the teeth with an acrylic appliance, the occlusal surfaces must be covered with acrylic. Usually this is a small amount, but if the freeway space is very small there may be contact of teeth, possibly causing a change in the balance of the stomatognathic system during swallowing, mastication, and speaking. There may even be contact in the rest position. Under these conditions, the dentist may choose to use a different method of temporary splinting, such as ligature wire. Permanent splinting of teeth requires considerable restoration and has many disadvantages which must be weighed against the possible advantages.²⁴

Splints are often used to protect the teeth from bruxism. This is an interim measure until diagnosis and effective treatment can be accomplished.

The term "bruxism" is interpreted differently by various authorities. Some consider bruxism as "... grinding of the teeth in other than chewing movements of the mandible, especially such movements performed during sleep."²⁸ Others specifically differentiate bruxism as being only nocturnal.⁹⁵ This differentiation is realistic because nocturnal tooth grinding differs considerably from tooth grinding in the wakened state. The noises and grating sounds of nocturnal tooth grinding are very often loud, crepitating sounds; significantly, the same sounds cannot be reproduced by the individual when awake. From

observations in their sleep laboratory, Reding, Rubright and Zimmerman consider this to be "... probably because in the waking state cortical inhibitions prevent the subject from exerting the full force of his muscles of mastication except when actually masticating. Furthermore, nocturnal and diurnal teeth-grinding occur in different stages of consciousness and differ sharply in their relationship with voluntary control." That the severe sounds may seem to almost be breaking the teeth can be confirmed by those who have shared a sleeping room with one who suffers from bruxism. Some individuals seek treatment for bruxism specifically because of the disturbance it causes to a sleeping partner.¹³⁶ Bruxism is not an uncommon problem. Studies have shown that it is present in 15% of the three to seventeen year age range; in 5% of the sixteen to thirty-six age range;⁹⁵ and in 22% of the twenty to fifty age range.⁷⁷

Damage done from bruxism is many-fold. There is a significant increase of tooth wear which is different from that of normal chewing. In bruxism the movement is primarily horizontal and in mastication primarily vertical; thus the cusps are shortened and the sulci more shallow.⁶⁰ There is controversy regarding the role bruxism plays in periodontal disease. Most agree that it is a contributory cause, but some deny that it can initiate the condition. A limited amount of bruxing may be beneficial to the periodontal tissues.¹³³ Meklas⁷⁷ states that bruxing may be stimulating to the supporting tissues in some patients.

Bruxism does appear to be involved with hypertonicity of the masticatory muscles. It becomes a question of which is primary, the bruxism or the hypertonicity of the muscles. Banasik and Laskin⁷ artificially produced bruxism in monkeys by electrical stimulation of the internal pterygoid and temporalis muscles. After a six-week period the muscles of mastication were studied by electromyography and showed the increase in frequency and amplitude of spike potentials comparable to clinically observed myofascial pain dysfunction syndrome. Jankelson⁶⁰ artificially induced bruxism by applying a thin film of acrylic cement on one or several contacting surfaces of the teeth. Bruxism immediately began in all ten of the individuals, who ranged from four to seventy-two years of age. This experimental production of bruxism correlates with the findings of many investigators which indict malocclusion as a major cause of bruxism.

The other major cause of bruxism is psychophysiology. Bruxism has been shown by electromyography and biofeedback methods^{107, 108, 109, 151} to relate to emotional stress. Rugh and Solberg^{107, 109}

found improvement of nocturnal bruxism as a result of biofeedback training and also by splint therapy. Neither treatment, although effective at the time, lasted. When the splints were removed, the previous level of bruxism returned.

Bite plane therapy is often designed to give a splinting action to the teeth for protection during bruxism. In some cases both an upper and a lower bite plane are used.⁹⁴ This is done to protect the teeth in both arches in severe bruxism. Care must be taken that there is adequate freeway space to prevent muscular contraction, which may develop as a result of splints interfering with the freeway space.¹⁴⁵ Splint therapy may be effective in reducing stimulation to the periodontal proprioceptors as the result of prematurity. In any event, the basic cause of the bruxism must be found and eliminated for a permanent cure.

Applied kinesiology investigation indicates that bruxism often correlates with poor lymphatic drainage. This is examined for with applied kinesiology's retrograde lymphatic technique. The patient is placed in a 20° retrograde position, with his head lower than his feet. Poor lymphatic drainage is indicated if a previously strong indicator muscle weakens but regains strength when the arms are placed above the head in 180° shoulder flexion. The hypothesis is that there is entrapment of the lymphatic vessels as they tortuously arc through the tissues to drain into the venous system near the subclavian and internal jugular vein. Structural correction, which usually requires attention to the pectoralis minor muscle, relieves the indication of this partial entrapment of lymphatic drainage. Bruxism is often eliminated by this correction (see

Volume I for complete discussion of retrograde lymphatic technique).

One possible cause of bruxism which has not been investigated is that the grinding action is a subconscious activity to mobilize a poorly functioning cranial primary respiratory mechanism. The mandible and the masticatory muscles are mobilizers for this mechanism. It can be observed using applied kinesiology methods that mandibular opening and closing movements, as well as clenching, can improve cranial primary respiratory function, at least temporarily. As has been noted earlier, mandibular action can be either beneficial or detrimental to skull activity. One of the factors indicating that bruxism may be an effort of the body to improve cranial function is that gum chewing often covers the clinical evidence of cranial faults. Under these circumstances, cranial faults appear shortly after a patient discontinues gum chewing. The cranial primary respiratory mechanism appears to be activated by many factors. Walking, with its continual stimulation and inhibition of muscles that insert into the cranium — such as the sternocleidomastoid and upper trapezius — seems to activate the cranium in normal individuals. The question arises whether nocturnal bruxism is an effort of the body to activate the skull when the general body is inactive during sleep.

It has been clinically observed that some people with chronic bruxism diminish or quit the activity as the stomatognathic system is corrected. Since many factors of muscular imbalance, cranial faults, and malocclusion are corrected, it is very difficult to determine which factor or factors cause the reduction of bruxism.

Equilibration

Occlusal equilibration has been defined by various authorities in many different ways. Generally it is considered a system of recontouring the tooth surfaces through selective grinding procedures to eliminate stressful contacts, equalize occlusal stress, load the teeth in their long axes, and prevent interference with normal mandibular and muscular action. Equilibration may be done to the natural dentition, restorations, or prostheses. It is a procedure requiring great skill and knowledge, and it must be correlated with the total stomatognathic system rather than simply tooth-to-tooth contact. In no case should it be taken lightly, especially when the natural dentition is involved, because selective grinding is an irreversible loss of tooth enamel.

Many equilibration systems are taught, and the procedure is usually modified by the operating dentist. Here we will briefly look at some of the methods used. Our primary objective is to put into perspective the very important role that occlusal equilibration plays in balancing the total stomatognathic system, and how it correlates with the applied kinesiology approach to normalizing the muscles.

Obviously it is important to determine who needs occlusal equilibration. We cannot consider this until it is decided how equilibration relates with centric relation and centric occlusion. It seems to be generally agreed that centric relation and centric occlusion should be coincident. The major question,

Occlusion

then, is where centric relation is considered to be. In Chapter 11, there was considerable discussion about whether centric relation should be with the condyle in the most retruded superior position, or in the neutral position of the fossa in the presence of balanced muscles. It seems that the only advantage of centric relation being in the most retruded superior position is that this is the terminal hinge axis; it is said to be located repeatedly, thus making it a standard reference point. If one chooses to equilibrate centric occlusion to centric relation in the most retruded superior position, then articulators can mount study models to move the upper and lower arches in the same arc as the mandible; thus study of occlusion and trial equilibration can be done on the study models.

Although this seems a very nice, convenient approach, the body in its intricacies simply does not work this way. Koski,⁶⁸ in a thorough study of the axis of opening movement of the mandible, concluded: "Mechanical constructions based on the hypothesis of a fixed axis are unsound. The great variability of mandibular movements and the location of the axis can hardly be reproduced by a man-made mechanical device."

One concept of making centric relation in the retruded superior position coincident with centric occlusion is that an individual will have one habitual path of closure to centric occlusion. This is supposed to occur because the engram of closing brings the mandibular arch into accurate intercuspation with the condyle in the most retruded superior position. On the Mandibular Kinesiograph, we nearly always see the habitual closure as having a deflexive posterior slide in those who have been equilibrated into retruded superior centric relation. The unequilibrated individual with apparently normal function most frequently has a slight anterior slide, with no lateral deflection when closing on the habitual path.

Additional arguments against the posterior hinge position are the detrimental effect to the disc³⁴ and the fact that the joint is forced to function at its limit of motion, which is an unsound physiologic position.³¹ The optimum choice for centric relation is with the condyle in the median area of the disc and fossa, as the mandible is held by the muscles of the mandibular sling when they are functioning normally. This position can be called physiologic centric relation.

Physiologic centric relation is not at the edge of the envelope of motion; thus when temporary structural or muscular changes take place, adaptation of the condylar position is possible. This is important in the role of the mandible and muscles of mastication for correction of cranial faults. The mandible must be able to move to adapt to temporary traumatic muscu-

lar imbalance and corrective engram muscular control, which change the various axes of mandibular movement.⁶⁸ Electromyography reveals different chewing patterns among individuals.¹³⁰ This variable is even present in individuals who have been equilibrated to a specific position, and it indicates the need of adaptation to meet the immediate demands of the system.

The final factor in favor of a physiologic centric relation coincident with centric occlusion is that nearly 90% of normally functioning individuals have this pattern.^{92, 101} If one were to believe that centric relation in the most retruded superior position should be coincident with centric occlusion, one would have to accept the concept that approximately 90% of apparently normally functioning individuals have deflexive malocclusion. If this is true, we need to know how malocclusion develops in an apparently normally functioning individual without symptoms. Furthermore, if this percentage of deflexive malocclusion is present in asymptomatic individuals, then one would have to accept the concept that deflexive malocclusion does not cause a problem. The argument that making centric occlusion coincident with the most retruded superior centric relation position, eliminating stress on the dentition and preserving it, simply does not coincide with physiologic observations of apparently normally functioning asymptomatic individuals.

The concept of the condyles in a physiologic centric relation coincident with centric occlusion in the presence of balanced muscles is not new. Stoll¹³¹ presented this general concept in the 1940s with the statement, "It is absolutely erroneous to assume that centric position is the position of the mandible in the most retruded position from which lateral movements can be made; that the condyle altogether guides the movements of the mandible; and that the condyle has a definite position in the fossa which is set at birth." The condyle must have a range of motion in which to function, and the musculature plays a predominant part in its posture and excursion.⁷¹

The discussion of occlusion and equilibration so far has assumed that there has been no loss of vertical dimension. It must always be remembered that there are three dimensions to consider in occlusion. In applied kinesiology it is best to consider vertical dimension after the other factors of stomatognathic function, such as muscular balance and cranial faults, have been considered and corrected, unless there is an extremely obvious loss of vertical dimension, such as loss of posterior support from absent teeth. The rest position and apparent requirements of vertical dimension change as muscular function changes with treatment.

There are many methods for determining muscular disturbance. Dawson²⁴ strongly supports palpating the muscles for tenderness, especially the pterygoid muscles. Electromyography, although not a method generally used on a clinical basis, reveals the muscular imbalance of malocclusion.^{83, 90} The Mandibular Kinesiograph measures mandibular movement. It is valuable in determining the balance of the muscular system in jaw movements, and it can be combined with electromyography for further evidence of muscular dysfunction. Applied kinesiology reveals considerable information about muscular function in mandibular movements; it also provides an ability to evaluate other muscles in the closed kinematic chain of the stomatognathic system and throughout the body.

It is important that the muscular imbalance almost universally present in malocclusion be corrected before equilibration is attempted. The discussion of centric occlusion coincident with physiologic centric relation in the presence of muscular balance sounds very fine, but remember our previous discussion of some major factors. It is not adequate to consider TMJ muscle balance only; any cranial faults and remote problems must be corrected prior to considering occlusion. These corrections should be done first because the next step is equilibration, an irreversible procedure. Muscular balance can often be completely obtained with applied kinesiology techniques. Even when it is necessary to use a bite plane to break down the engram, applied kinesiology techniques reduce the time needed for the therapy. (AK examination and treatment are presented in Chapter 14.)

The need for equilibration is indicated by standard dental procedures. Methods of observing for deflective slide on entering centric occlusion have been discussed. Most often the prematurities are marked with marking ribbon, paste, or occlusal indicator wax. Applied kinesiology examination also indicates need for equilibration. Jankelson⁶¹ notes, "Before proceeding with the clinical adjustment of the occlusion, the dentist should have a clear understanding of what has happened to disrupt it and what is required to restore it to a favorable functional state." This is extremely important and indicates a need for any physician — dentist or not — to understand the interactions of the stomatognathic system and its interactions with the total body. Malocclusion does not develop without a reason. Simply equilibrating an individual's occlusion without understanding why malocclusion developed is often treating the symptom without adequate attention given to the primary cause.

With structure balanced, registering prematuri-

ties is the next step. The condyles are in the physiologic centric relation, with the mandible held in position by the balanced mandibular sling. The mandibular arch is registered to the maxillary arch with the patient in an upright, head level position. There is mild physiologic resistance to the normal closure from the natural upright posture. Ricketts⁹⁹ states, "It results in a centered-condyle position away from the posterior terminal condyle location but not sufficiently forward to be on the slope of the eminence." With marking ribbon or occlusal indicator wax held between the teeth, prematurities are located by lightly tapping the teeth together.

The reason for the patient sitting upright with his head level during registration of centric occlusion is to ensure that the muscles in the closed kinematic chain of the stomatognathic system will be balanced. McLean et al.⁷⁶ demonstrated that various postures changed all three dimensions of the rest position, but the engram controlling mandibular closing brought the teeth into accurate centric occlusion; thus when the occlusion is registered and equilibrated to balanced muscles in the seated position, the closing to centric occlusion will be accurate. Another important aspect of the upright position is so the patient's head will not be against a headrest; this prevents distortion of the cranium. Under these circumstances — no cranial faults present, balanced muscles in the total stomatognathic system, and no remote factors adversely influencing the system — accurate registration can be made.

It is relatively simple to register malocclusion. What requires a high level of expertise and skill is balancing all aspects of the stomatognathic system and remote factors prior to evaluating for malocclusion and, finally, correctly equilibrating the patient with selective grinding.

Because Silverman¹²² observed that the skull had flexibility which could change the occlusion, he devised a system for bringing the skull into normal balance prior to marking the occlusion for equilibration. It has been pointed out many times throughout this text that external forces applied to the skull can potentially distort it. Since equilibration has developed to a very fine procedure, it seems reasonable that Silverman's precautions can enhance the quality of the final result. (1) The head should be unsupported. (2) The saliva ejector should not be used to avoid pressure on the teeth. (3) Wide opening of the mouth should be avoided. (4) The articulating paper (marking ribbon), paste, or occlusal indicator wax is placed over the teeth, and the patient is asked to close his mouth until the teeth are almost touching. (5) The patient is told to inhale and exhale as deeply as possible, at least three times, and then tap or make

Occlusion

excursive movements for marking.

Jankelson has developed a system for registering the rest position and occlusion, using neuromuscular principles.⁶⁴ The system uses transcutaneous electrical neural stimulation (TENS), produced by the Myo-monitor,⁸⁵ to cranial nerves V and VII. The instrument provides pulsing to the masticatory and facial muscles at a rate of 40 pulses per minute. The impulse is 500 microseconds in duration and continuously variable in amplitude from 0 to 25 milliamps maximum. The stimulation of the muscles is done for thirty to forty-five minutes; it is said to relax the musculature to its resting length. The effect of the stimulation can be observed on the Mandibular Kinesiograph. After pulsing there is often an increased freeway space, and the mandible is more stable in the rest position. This is especially true if there is considerable instability of the rest position prior to treatment. In the event that the rest position does not stabilize after Myo-monitor treatment, Jankelson recommends pre-medication with Valium.

The Myo-monitor has many uses in dentistry. After muscle relaxation, the instrument can be used for occlusal registration for mounting study models, registering prematurity, determining bite plane requirements, and molding denture borders.

In some respects the philosophy behind the use of the Myo-monitor parallels that of applied kinesiology; in others ways it differs significantly. Both consider that the condyle should be located in the fossa in the center of muscular balance, and that muscle balance is a prerequisite in determining prematurities, rest position, and occlusal registration. In the Myo-monitor philosophy, the muscles of the face and mastication are said to be brought to their relaxed state by TENS, whereas in applied kinesiology philosophy an attempt is made to determine why the muscles are not in their relaxed state and then correct that factor. Both systems register prematurity by muscle contraction, without the examiner influencing mandibular movement in any way. The muscle contraction used in applied kinesiology is provided by the patient's effort. In the AK system care must be taken that there is not a residual engram improperly elevating the mandible into an imbalanced position. This requires that all the factors presented in Chapter 14 be evaluated and corrected properly. In the case of a deeply established engram of closing, it may be necessary to use a bite plane to allow the engram to subside. In the Myo-monitor philosophy, the muscle contraction is provided by TENS. Since the stimulation is through cranial nerves V and VII, all muscles supplied by these nerves contract.⁶² The question arises as to

whether general stimulation of cranial nerves V and VII activates the mandibular closers toward occlusion in the same way that proper volitional control would. It seems that the external stimulation precludes some or all of the neurologic control of muscle synergism. Both methods of registering prematurity appear to have a questionable drawback, yet both methods are far superior for registering occlusion compared with the retruded superior centric relation position.

Other uses of the Myo-monitor have no comparison or parallel with applied kinesiology techniques. It is used to elevate the mandible into a recording medium to produce an impression for mounting study models. The repeated muscular contraction produced by the Myo-monitor brings the mandible up to the pre-determined rest position. The recording medium (Myo-print)⁸⁵ is a plastic that hardens like acrylic. When the impression is properly made, the substance keeps its accuracy until it cures to the hardened state, and then continues to keep it indefinitely. The important factor about this method of occlusal registration is that it uses the muscle trajectory from the rest position to make the impression. This places the condyles in the fossa in what Jankelson calls myo-centric occlusion, giving compatibility of the occlusion with physiologic centric relation.

No studies have been reported to determine if the occlusal registration as obtained by the Myo-monitor is compatible with the movement of the mandible toward centric occlusion when the muscles are functioning normally and under volitional control. The stimulation of cranial nerves V and VII by TENS would seem to cause maximum contraction in all muscles, and fail to obtain the same muscle synergism that volitional control would.

Techniques have been developed for taking denture impressions and relining old dentures. The muscular contraction caused by the Myo-monitor is used to mold denture borders. It is said to provide a better stabilization of the final prosthesis for the edentulous patient.

A major consideration in equilibration is that it be done in the presence of normal function of the total stomatognathic system. It seems important to repeat Perry's⁸⁹ statement, "The evaluation of muscle function may some day be every bit as important as observing the intercusping of the opposing teeth in occlusion." Balancing function prior to occlusal equilibration is a primary factor for optimum results and the prevention of iatrogenic conditions.

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Chapter 13

Hyoid Muscles and Function

Introduction

The muscles associated with the hyoid have received less attention in the literature than other muscles of the stomatognathic system. They are usually studied as the supra- and infrahyoid muscle groups, and will initially be considered that way here. Although this is a convenient anatomical grouping for study, it tends to isolate the function of these muscles from the rest of the stomatognathic system in the student's mind. These muscles — a very important part of the closed kinematic chain of the stomatognathic system — have intricate and complicated interactions with mandibular movement, phonation, deglutition, and balancing activity within the

closed kinematic chain.^{3, 11} In addition to the important role these muscles play in movement and stabilization of structure, they appear to have a major role in helping regulate equilibrium and balance between the two sides of the body. This activity is from the muscle proprioceptors, and it also appears to relate with the balance of the electromagnetic fields of the body.

This chapter will consider the anatomy of the hyoid and its muscular suspension, the apparent role of the hyoid as a gyroscopic-type balancing system,⁷ and applied kinesiology examination and treatment of hyoid muscle imbalance.⁷

Hyoid Bone Anatomy^{9, 15, 28}

The hyoid bone is U-shaped and has no direct articulation with another bone. It is suspended by muscles and ligaments and moves within this sling-type arrangement. It has a body and two greater and two lesser cornua.

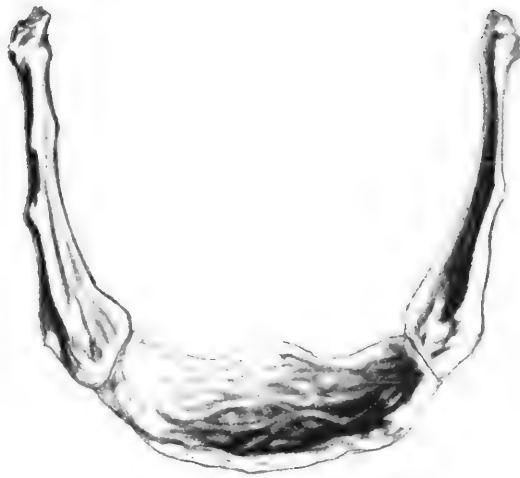
The body of the hyoid bone is the central part. The convex anterior surface is directed anteriorly and superiorly. The upper part is marked by a ridge, and often there is a vertical median ridge dividing the body into lateral halves. The posterior surface is concave, smooth, and faces inferiorly and posteriorly. It is separated from the epiglottis by the thyrohyoid membrane and a quantity of loose areolar connective tissue, with a bursa intervening between the bone and the membrane.

The geniohyoid muscle inserts into the greater

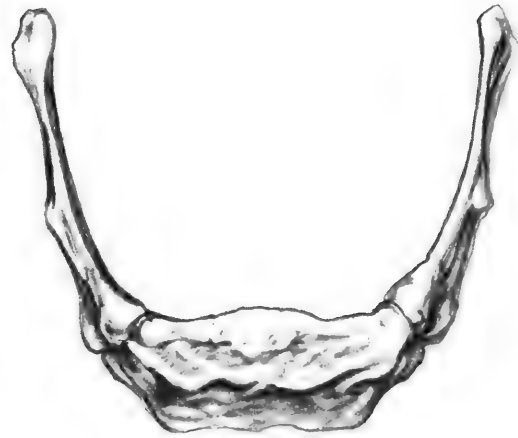
portion of the anterior surface of the body. A portion of the hyoglossus inserts into the body lateral to the geniohyoid. The mylohyoid, sternohyoid, and omohyoid insert inferiorly into the transverse ridge. Into the inferior border insert the sternohyoid medially and the omohyoid laterally. Attached to the superior border of the hyoid are the thyrohyoid membrane and some aponeurotic fibers of the genioglossus.

The greater cornua project posteriorly from both lateral edges of the body. They are made up of flattened bone diminishing in size to end in a tubercle, to which the lateral hyoid ligament attaches. Along the entire length of the roughened superior surface originate the hyoglossus and the middle constrictor muscles. The digastric — through an intermediate tendon — and stylohyoid muscles insert

Hyoid Muscles and Function



13—1. Inferior view of hyoid bone.



13—2. Superior view of hyoid bone.

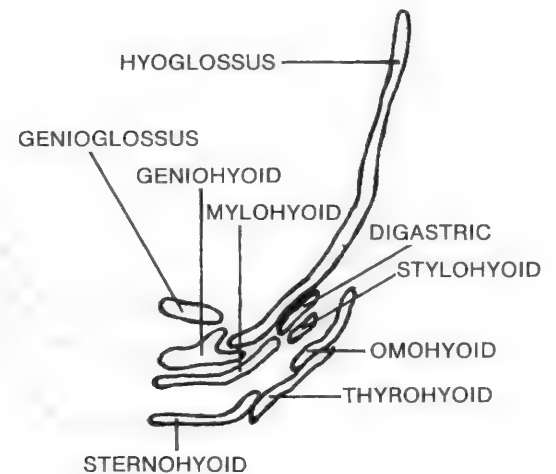


13—3. Inferior view of muscle insertion to the hyoid bone.

near where the cornua arise from the body. The thyrohyoid muscle inserts into the anterior lateral border.

The lesser cornua are two small conical eminences. Their bases attach at the angle where the greater cornua attach to the body on both sides. The articulation is by fibrous tissue, which may become osseous in later life. The stylohyoid ligament is attached to the cornu, and the chondroglossus muscle arises from the medial side of its base. The stylohyoid ligament is a fibrous cord from the lesser cornu of the hyoid bone to the styloid processes of the temporal bones.

The hyoid is connected to the thyroid cartilage by the thyrohyoid membrane and a middle and two lateral thyrohyoid ligaments. The thyrohyoid membrane attaches to the posterior superior surface of the body and greater cornua of the hyoid, passing



13—4. Superior view of muscle insertion to the hyoid bone.

downward to attach to the superior border of the thyroid cartilage and to the front of its superior cornua. The central part of this broad fibroelastic membrane is called the median thyrohyoid ligament. The lateral thyrohyoid ligaments are on the lateral posterior borders of the thyrohyoid membrane; they attach to the hyoid at the greater cornua, passing to the tip of the superior cornua of the thyroid cartilage.

The hyoid can be palpated immediately above the thyroid cartilage. The anterior superior portion of the thyroid cartilage can be located by palpating the thyroid notch anteriorly. The hyoid bone is best palpated with the thumb on one side and the index finger on the other side. It is located in the receding angle between the chin and anterior part of the neck on the level of the 4th cervical vertebra. Its greater cornua extend back on a level with the angle of the mandible.



13—5. The hyoid is immediately above the thyroid cartilage. Palpate for the thyroid notch. Move up to the hyoid and locate its superior border.

Muscles of the Hyoid

It is important in applied kinesiology to be able to visualize the location of the hyoid muscles, as well as to know their action on the hyoid in various body functions, because applied kinesiology examination requires moving the hyoid to stretch the musculature. The hyoid is moved to stretch the hyoid muscles during challenge. The most accurate evaluation is when the hyoid movement is in alignment with the fibers of a specific muscle. When the hyoid is moved in a general direction — laterally, superiorly, inferiorly, etc. — it is necessary to know which

muscles are being stretched.

Learning the origin and insertion of each hyoid muscle is basically very easy. In many cases the name of the muscle is that of its origin and insertion.

The format in which the muscles of the hyoid are presented gives their origin, insertion, description, action, and nerve supply.^{9, 14, 15, 20, 27, 28} How to best locate each muscle is discussed in the section on description, since it is necessary to therapy localize and treat the muscle during examination and therapy.

SUPRAHYOID MUSCLES

DIGASTRIC

The digastric muscle is different from other hyoid muscles because it has two bellies with an intermediate rounded tendon surrounded by a fibrous loop which connects the bellies to the hyoid.

Posterior Belly

Origin: Mastoid notch (digastric fossa) on the posterior medial aspect of the mastoid process of the temporal bone.

Insertion: Into the intermediate tendon, which is attached to the lateral aspect of the hyoid body and greater cornu by a fibrous loop.

Anterior Belly

Origin: Digastric fossa of the mandible, which is a depression on the inner side of the inferior border of the mandible close to the symphysis menti.

Insertion: Into the intermediate tendon, which is attached to the hyoid as noted above. The intermediate tendon perforates the stylohyoid muscle.

Description: The posterior belly of the digastric is longer than the anterior. It angles inferiorly, anteriorly, and medially from the temporal bone toward the hyoid. The anterior belly is broader at its origin, narrowing as it approaches the intermediate tendon.

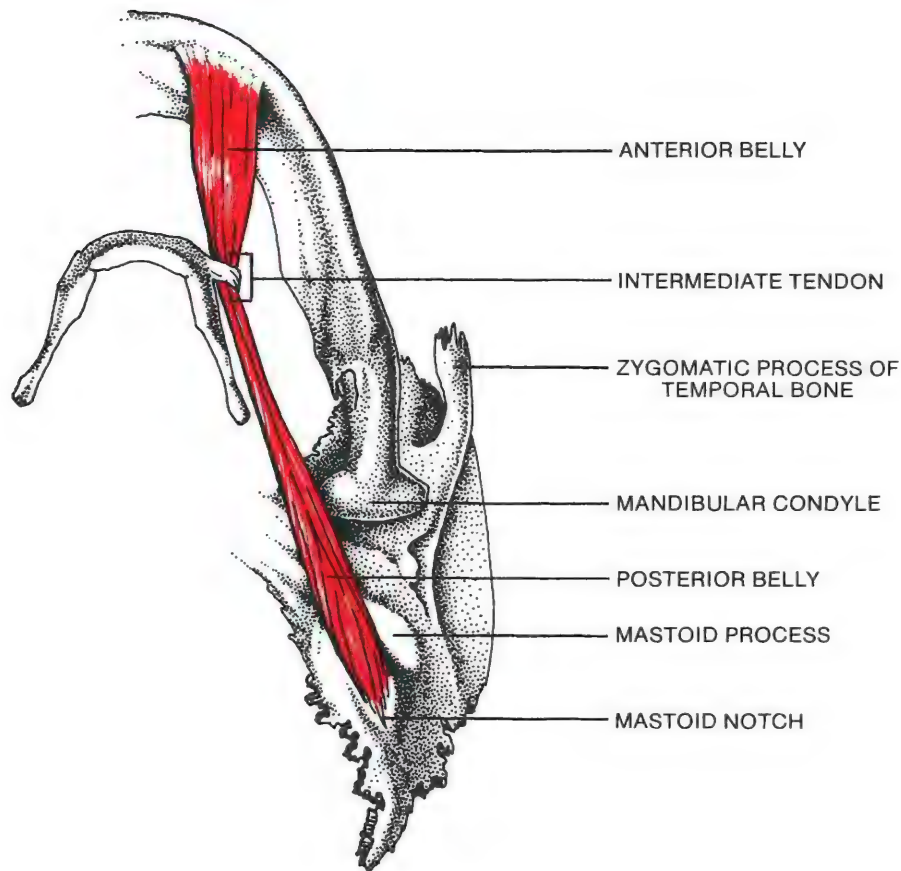
Location of the posterior belly of the digastric for therapy localization and palpation is along a line from the lower anterior aspect of the mastoid process to the upper border of the body of the hyoid. The anterior belly is relatively accessible from the inferior aspect of the mandible. The origin is approximately

2 cm wide, with the medial border of the muscle close to the symphysis menti. The muscle narrows as it approaches the hyoid, and it courses somewhat laterally.

Action: In general, the anterior belly of the digastric is a jaw-opening muscle. Acting together, the digastric bellies can elevate the hyoid bone.

Electromyography appears to clarify the question of what muscles act in mandibular depression. Moyers¹⁶ found that the external pterygoid muscle acts first on jaw opening, but it is quickly followed by contraction of the anterior belly of the digastric muscle. When mandibular opening is forced, the anterior belly has increased contraction which begins earlier in the movement. Mandibular depression cannot be carried to its final normal completion without contraction of the anterior belly of the digastric muscle. Moyers¹⁶ concludes “. . . that the external pterygoid is more responsible for the initiation of the mandibular depression but that the digastric plays an important role in completing the movement.”

The right and left digastric muscles always function together, as observed on electromyography.²⁹ Although the muscles function together bilaterally, the anterior and posterior bellies have independent activities. Munro¹⁸ found that when the periodontal ligaments are stimulated during mastication, the posterior belly contracts without activity in the anterior belly. This study seems to indicate that the intermediate tendon does not slide freely through its fibrous sling. The suprahyoid muscle group is important in stabilizing the mandible during all movements; this achieves a more delicately adjusted action.¹⁶



13—6. *Inferior view of the anterior and posterior bellies of the digastric.*

Stimulation of the periodontal ligament receptors by occlusion or tapping on a tooth causes inhibition of the mandibular elevators; this is called the jaw-opening reflex,^{1, 10, 17, 22} discussed in Chapter 9. Griffin and Munro¹⁰ demonstrated by electromyography that there is activation of the anterior belly of the digastric muscle during the silent period of the mandibular elevators following stimulation to the periodontal ligament receptors. This indicates that the digastric provides a protective mechanism to relieve stress on the dentition during chewing. Their finding was not confirmed by Beaudreau et al.;² however, Beaudreau's group did influence the muscle's activity by tapping on a tooth to stimulate the periodontal ligament receptors. This gives evidence that at least this muscle of the hyoid group is directly influenced by the periodontal ligament receptors; probably all the muscles of the superior and inferior hyoid groups are influenced in some way by them.

Basmajian¹ summarizes the work of several investigators concerning the activity of the digastric

muscles in their general function, such as coughing, breathing, chewing, and swallowing. All of these activities strongly recruit the activity of the digastric muscles.

Palpation of the posterior belly is along a line beginning at the mastoid notch to a point at the superior aspect of the hyoid. The patient's head should be slightly extended and laterally flexed to elevate the angle of the mandible, uncovering the posterior belly of the digastric. The anterior belly is palpated in the area from slightly above the hyoid to the digastric fossa. Neuromuscular spindle cell treatment to both bellies requires probing somewhat into tissue to contact the muscle.

Nerve Supply:

Anterior belly: Mylohyoid branch of inferior alveolar branch of the mandibular division of the trigeminal nerve.

Posterior belly: Branch of the facial nerve.

Hyoid Muscles and Function

STYLOHYOID

Origin: Posterior lateral surface of the styloid process, near the base.

Insertion: Body of the hyoid at the junction with the greater cornu above the omohyoid.

Description: A slender muscle which courses anteriorly and inferiorly to the posterior belly of the digastric. The tendon of the digastric passes through the stylohyoid near its insertion.

Action: Moves the hyoid superiorly and posteriorly toward the styloid process. It stabilizes the hyoid posteriorly during action of tongue muscles originating from the hyoid. The stylohyoid, along with the geniohyoid, determines the length of the floor of the mouth.¹²

The stylohyoid is found on a line approximately from the external auditory meatus to the body of the hyoid. To therapy localize or treat the muscle, the head and neck must be in extension, or the head

turned away from the side of investigation. This head position is necessary; otherwise the angle of the mandible covers the muscle.

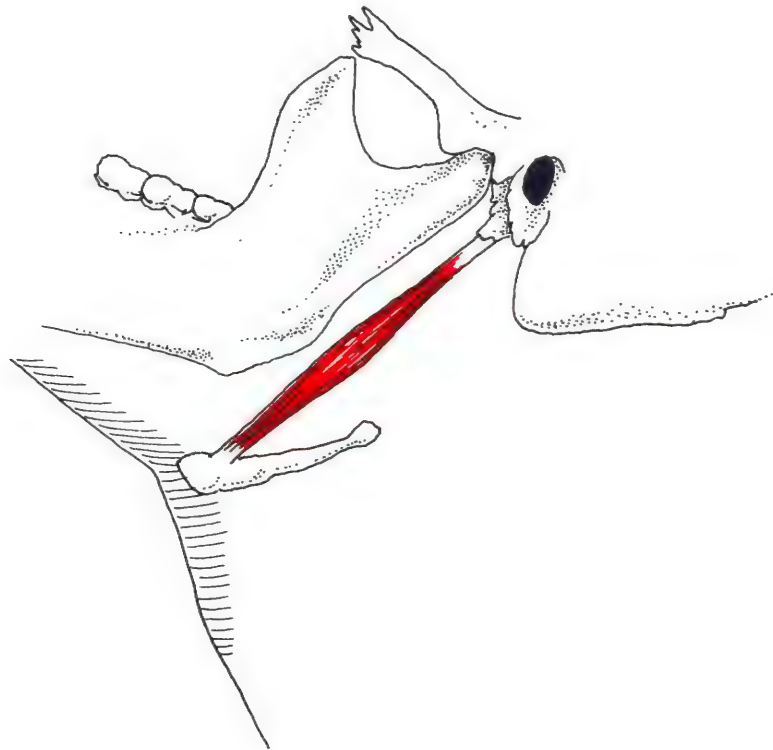
Nerve Supply: Facial nerve.

MYLOHYOID

Origin: From the mylohyoid line of the mandible which extends from the symphysis menti to the last molar.

Insertion: Into the body of the hyoid. The median edge of the muscle inserts into a fibrous raphe which extends from the symphysis menti to the body of the hyoid; thus the bilateral muscles join at an angle along their entire median length.

Description: The muscle is a flat, triangular sheet located adjacent and superior to the anterior belly of the digastric. The joined bilateral muscles form the muscular floor of the oral cavity.



13—7. Stylohyoid muscle. The head is extended on the cervical spine to raise the angle of the mandible away from the muscle.

GENIOHYOID

Origin: Inferior genial tubercle on the posterior surface of the symphysis menti.

Insertion: Anterior surface of the body of the hyoid.

Description: A narrow muscle which lies deep (superior) to the medial border of the mylohyoid. The medial borders of the bilateral muscles lie in contact with each other.

The geniohyoid, being deep (superior) to the mylohyoid, is more difficult to contact in the anterior area. It is questionable whether the mylohyoid or the geniohyoid is being treated. Its location is on a line from the symphysis menti to the body of the hyoid.

Action: Pulls the hyoid superiorly and anteriorly, or, if the hyoid is the fixed origin, pulls the mandible posteriorly and inferiorly. It is active in anterior movement of the tongue. Determines the length of the floor of the mouth with the stylohyoid muscle.¹²

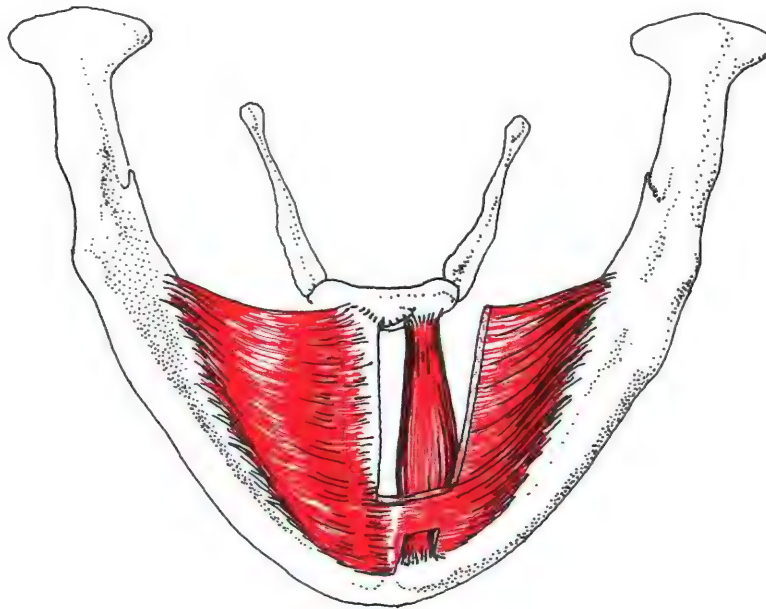
Nerve Supply: Branch of the 1st cervical nerve by way of the hypoglossal nerve.

The fibers of the mylohyoid are angled from the origin on the mandible inferiorly, medially, and somewhat posteriorly. They are located relatively easily for therapy localization and treatment in the anterior area of the mandible, and are relatively deep in the area of their posterior origin. This muscle is often more easily contacted if the patient is asked to force his tongue deep into the lower floor of the mouth. This also stabilizes the mylohyoid against the pressure of apparent proprioceptive treatment.

Action: Raises the hyoid, base of the tongue, and floor of the mouth. It is active in mastication, deglutition, sucking, and blowing.

Because the bilateral muscles form the broad, flat muscular floor of the oral cavity, these muscles were previously known as the oral diaphragm.⁴

Nerve Supply: Mylohyoid branch from the inferior alveolar branch of the mandibular division of the trigeminal nerve.



13—8. Mylohyoid and geniohyoid muscles.

INFRAHYOID MUSCLES

STERNOHYOID

Origin: Posterior surface of the medial end of the clavicle, posterior sternoclavicular ligament, and the upper and posterior parts of the manubrium.

Insertion: Inferior border of the body of the hyoid.

Description: A thin, narrow muscle which courses from its origin superiorly and medially, often joining with its fellow toward the superior portion.

This muscle is relatively easy to palpate, as it courses just lateral to the median line and along the anterolateral aspect of the larynx. As it passes over the superior border of the thyroid cartilage, the fibers are directed somewhat medially to insert on the body of the hyoid.

It is suspected in applied kinesiology that the afferent supply from the neuromuscular proprioceptors of this muscle, along with that of the sternothyroid and thyrohyoid, contributes information about the anterior-posterior position of the body-on-head organization to the central nervous system. They are important muscles to consider when the A-P curves are disturbed.

Action: Stabilizes and draws the hyoid inferiorly. It is active in mastication, speech, and deglutition.

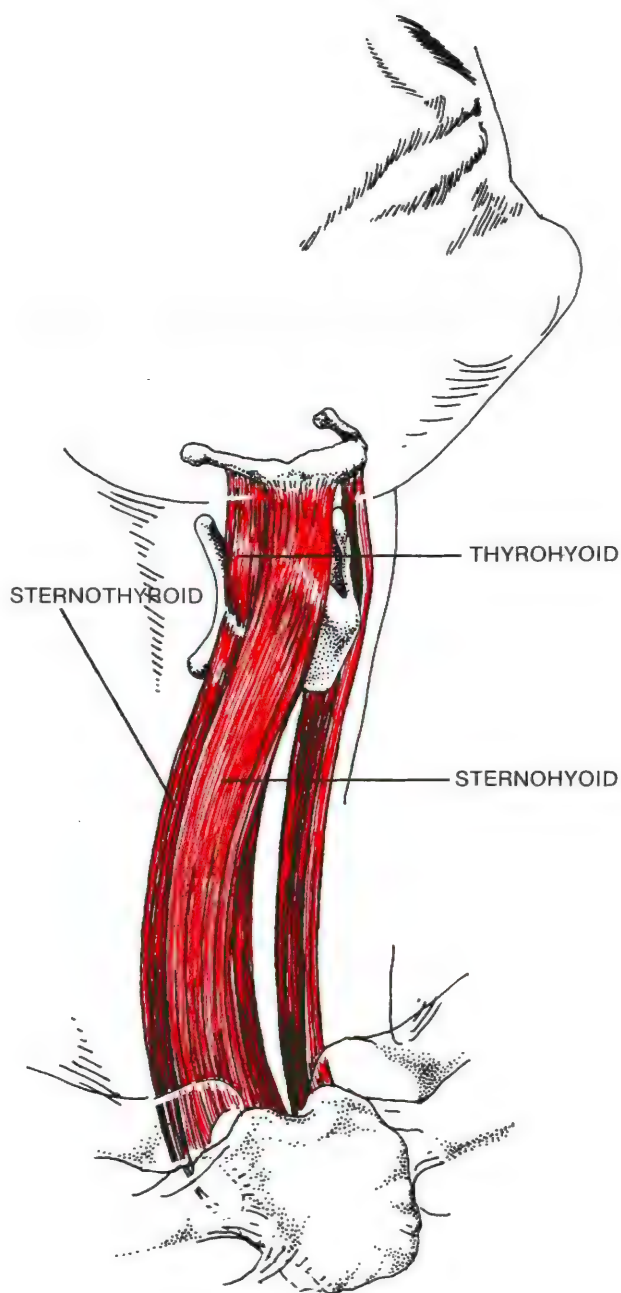
Nerve Supply: Branch of the ansa cervicalis with branches from C1, 2, and 3.

STERNOTHYROID

Origin: Edge of the cartilage of the 1st rib and from the posterior surface of the manubrium below the sternohyoid origin.

Insertion: Oblique line on the lamina of the thyroid cartilage.

Description: Shorter and wider than the sternohyoid muscle. It is situated deep to the sternohyoid. This muscle does not directly attach to the hyoid, but it is important in inferior stabilization of the structure. Palpation and therapy localization of this muscle are very similar to those of the sternohyoid. When the hyoid is challenged superiorly in applied kinesiology technique, it is quite possible this muscle is also being stretched. The applied kinesiology indication to evaluate it is when swallowing or phonation added to AK examination technique gives positive results, but the examination does not without the phonation or swallowing.



13—9. Anterolateral view of sternohyoid, sternothyroid, and thyrohyoid.

Action: Draws the larynx inferiorly after it has been elevated in swallowing or in vocal movements.

Nerve Supply: Branches from the ansa cervicalis, C1, 2, and 3.

Hyoid Muscles and Function

THYROHYOID

Origin: Oblique line on the lamina of the thyroid cartilage.

Insertion: Lower border of the greater cornu and adjacent part of the body of the hyoid.

Description: A small, quadrilateral muscle which could be considered as a continuation of the sternohyoid. It lies deep and somewhat lateral to the sternohyoid. Like the sternohyoid muscle, it is stretched when the hyoid is challenged superiorly. Its possible involvement is indicated when phonation

and swallowing are added to another examination procedure, which is then found positive after previously being negative.

Action: Draws the hyoid inferiorly or, if it is the fixed origin, draws the thyroid cartilage superiorly.

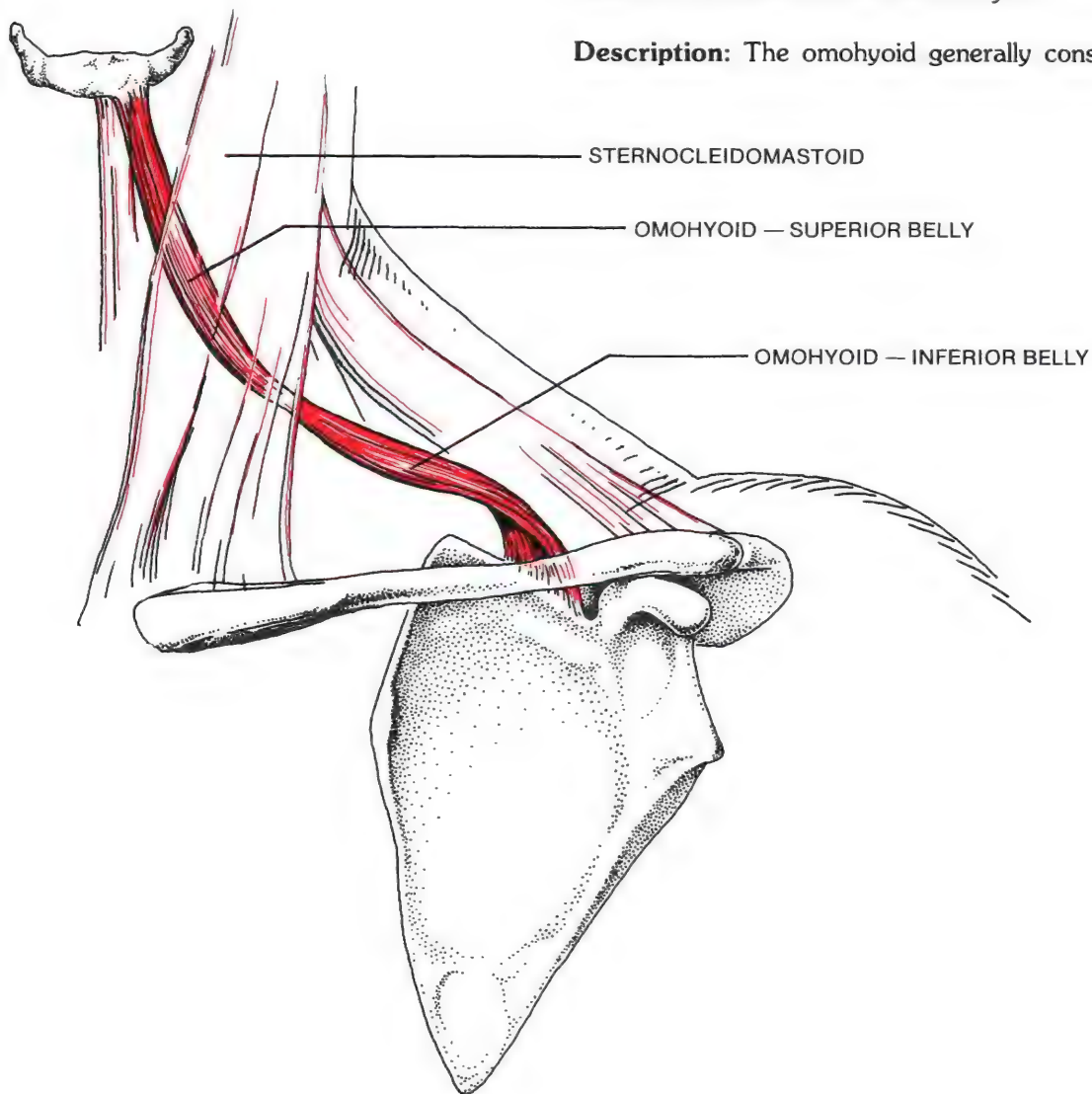
Nerve Supply: Branch of the ansa cervicalis with branches from C1 and possibly C2.

OMOHYOID

Origin: From the upper border of the scapula near the scapular notch.

Insertion: Inferior border of the body of the hyoid, lateral to the insertion of the sternohyoid.

Description: The omohyoid generally consists of



13—10. Omohyoid muscle.

Hyoid Muscles and Function

two bellies which are united by an intermediate tendon deep under the sternocleidomastoid muscle. The lower belly is a flat, narrow band which angles anteriorly, medially, and somewhat superiorly from the scapula to the lower portion of the neck. As it passes under the sternocleidomastoid at the point of its tendinous junction with the superior belly, the muscle changes direction to run almost vertically, close to the sternohyoid muscle, to insert into the body of the hyoid bone. The change of direction is from deep cervical fascia which ensheaths the muscle at the angular area, attaching it to the clavicle and 1st rib below.

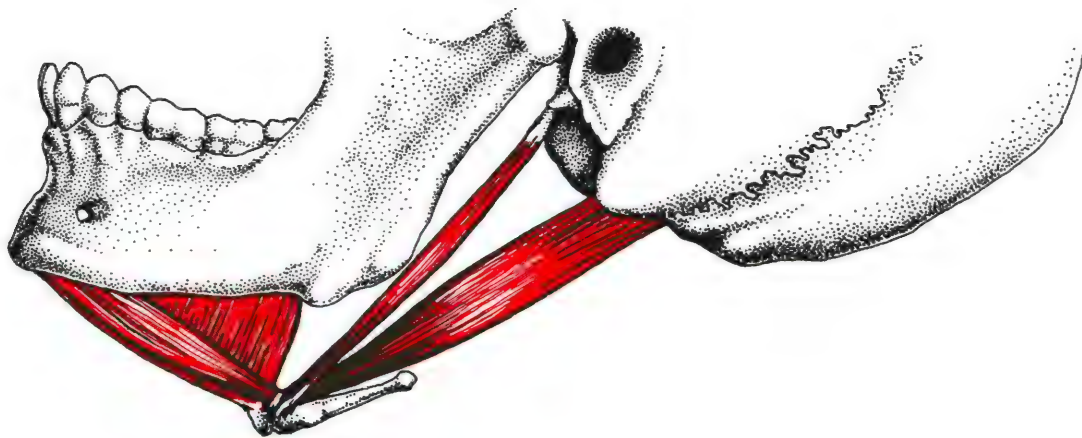
The superior belly is palpated and therapy localized as it emerges from under the lower aspect of the sternocleidomastoid. It is located on a line from that point to the hyoid, mostly adjacent to the

sternohyoid muscle. The inferior belly is palpated as it emerges from under the lower portion of the sternocleidomastoid and courses almost horizontally to the scapula.

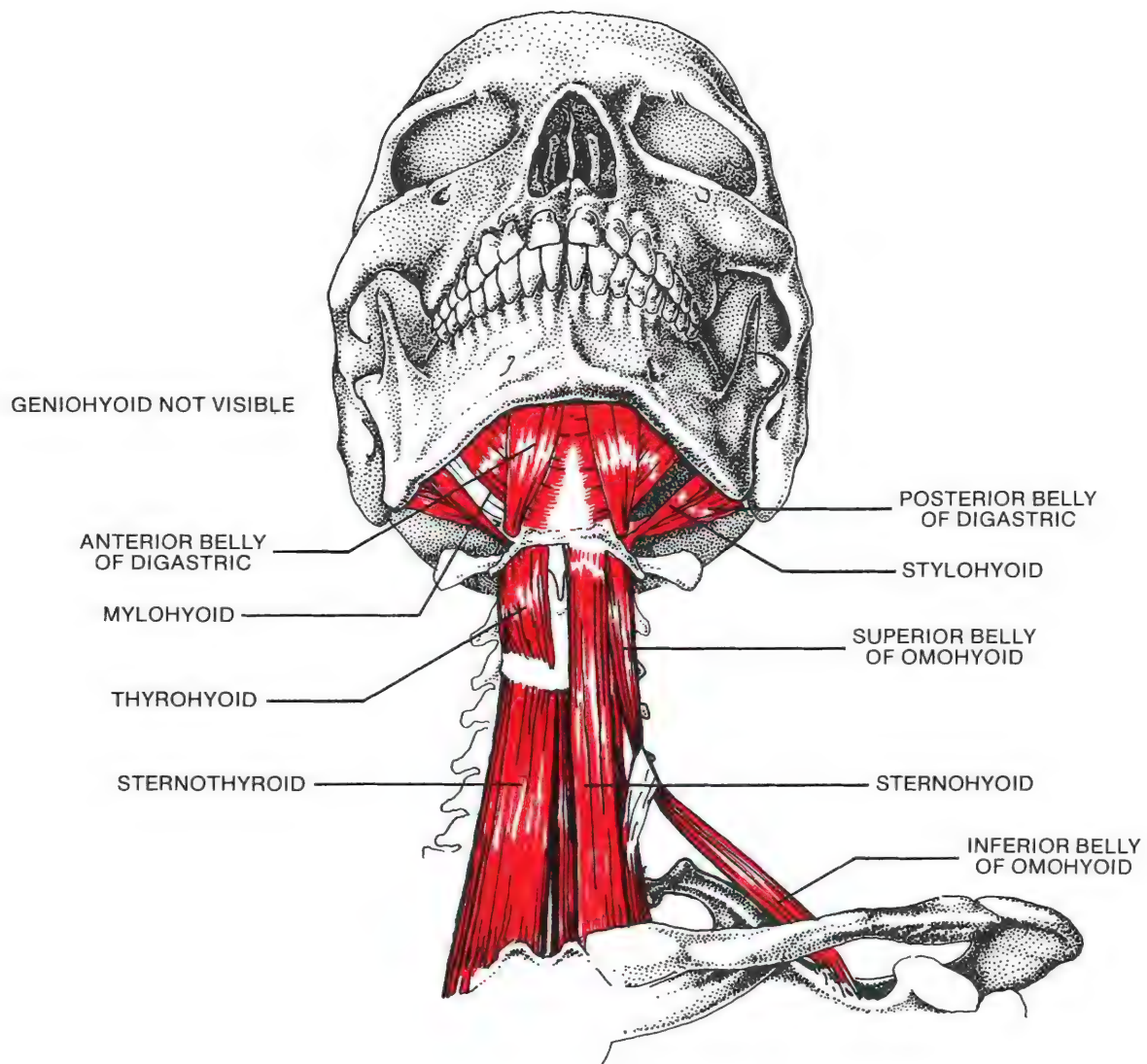
It is suspected in applied kinesiology that the afferent supply from the neuromuscular proprioceptors of this muscle is very important in the lateral orientation of the head and neck complex with the shoulder girdle. Treatment to the neuromuscular spindle cell often changes the orientation of the body-on-head organization.

Action: Moves the hyoid and larynx inferiorly. Steadies the hyoid from the inferior, and is suspected to be important in inspiration.

Nerve Supply: Ansa cervicalis, C1, 2, and 3.



13—11. Suprahyoid muscles.



13—12. Hyoid muscles.

Hyoid Muscular Balance

It is unfortunate that there is not more knowledge about the individual function of the hyoid muscles. Limited electromyographic studies have been done, but to individually study these small muscles it is necessary to use needle or fine wire electrodes which are problematic, to say the least, to insert into the muscles. In general, anatomy texts tend to group the hyoid muscles according to their action. The superior hyoid muscles are usually considered elevators, while the inferior ones are believed to draw the hyoid down or stabilize it. Last¹² points out that muscles anatomically in contact do not necessarily share a common action. He states, "In a functional sense, there is no such group as the suprahyoid muscles." In discussing the individual action of the suprahyoid muscles, he states that the digastric muscle helps depress the mandible, the mylohyoid elevates the floor of the mouth, and the geniohyoid and stylohyoid determine the length of the floor of the mouth.

It is generally agreed that the anterior belly of the digastric is active in jaw opening,^{1, 16, 29} and that the muscle is primarily important in maximum jaw opening. There have been no electromyographic studies found regarding the role of other hyoid muscles acting as muscle couples with the anterior belly of the digastric. It seems obvious that since the hyoid is fixed primarily by muscular action, it is necessary for other muscles to act as fixators for the anterior belly of the digastric to effectively function in jaw opening. These muscles would probably include the posterior belly of the digastric and the stylohyoid, to fix the hyoid posteriorly. The infrahyoid muscles, such as the sternohyoid and omohyoid, are probably required to stabilize the hyoid inferiorly. This hypothesis of muscle-couple function in hyoid activity is supported by clinical evidence in applied kinesiology which denotes improved temporomandibular joint function following therapeutic efforts to the hyoid musculature.

The interaction of the hyoid muscles in simple jaw opening, as described above, is relatively simple when compared with the more complex activities of deglutition, phonation, and mastication, especially when any of these are combined with head movement in flexion, extension, side-bending, or rotation. Since the total complex of the hyoid muscles is part of the closed kinematic chain of the stomatognathic system, it must adapt as the cervical extensors or any other muscles in the mechanism contract or relax. This constant need to adapt and still function correctly in the complex activities of phonation, deglutition, and mastication truly makes this a highly organized system of muscle function.

During mastication the anterior belly of the digastric is active as a jaw-opening muscle, probably along with the muscle couples discussed above. It is difficult to know exactly how the hyoid muscles function during the various activities. Most studies have shown statistically reproducible patterns per individual, but great variance between subjects. It appears that a specific engram of function develops for each individual, depending upon the type of occlusion, temporomandibular joint function, style of phonation, and habitual swallowing patterns. These patterns of neuromuscular function may be influenced by various types of structural dysfunction. The hyoid muscles receive their nerve supply from cranial nerves V and VII and the ansa cervicalis (C1, 2, and 3). Applied kinesiology clinical observations indicate that hyoid muscle function can be disturbed by cranial faults or upper cervical subluxations. Treatment to the neuromuscular spindle cell and Golgi tendon organ is another clinically effective therapeutic approach in applied kinesiology.

The hyoid, held in position by its muscular complex, has been compared with a gyroscopic feedback system by Goodheart.⁷ This was discussed on page 278 when considering the activity of the head-to-shoulder girdle musculature. In a somewhat related deliberation he then considered the neuromuscular balance of the hyoid to be involved in communication between the two sides of the body.⁸ This occurred as a result of observing the effect of two-handed therapy localization in the applied kinesiology ligament interlink technique (see Volume I). When the hyoid was moved laterally, the positive two-handed therapy localization was interrupted. Holding the hyoid laterally is part of the treatment when using the ligament interlink technique. It is unknown what neurologic relation may be present in this activity, but the technique is clinically effective.

There is further clinical evidence that the neuromuscular balance of the hyoid relates with organization between the two sides of the body. Goodheart⁸ observed that subjects reacted differently to manual muscle testing during right brain activity (humming) and left brain activity (saying the multiplication tables), depending on whether the muscles of the hyoid were balanced or not. Weakening of remote muscles on either right or left brain activity is often abolished when the hyoid muscles are balanced.

Another unusual observation in applied kinesiology is that when there is a lateral occipital subluxation, an indicator muscle will weaken when the patient extends his tongue from his mouth and points it in the direction of the lateral subluxation.

This is thought to relate to the neuromuscular system of hyoid suspension as it organizes with head leveling, which may be disturbed by the occipital subluxation. The lateral direction of tongue extension is consistent with challenge and other evidence indicating an occipital subluxation on that side.

Under certain conditions body function changes when the electromagnetic fields around it change. (This subject is discussed in Volume III of this series.) An unusual observation of the body's reaction to magnetism will be briefly mentioned here in relation to hyoid neuromuscular balance. Goodheart⁷ noticed that when there is poor neuromuscular balance of the hyoid muscles, as observed by applied kinesiology methods, any muscle of the body will weaken with manual muscle testing if a 3,000 gauss magnet is held lateral to the hyoid on one side, and another magnet is held on the other side with opposite poles next to the skin. It makes no difference whether the north pole is held next to the skin on the left or right

side, just so the poles are opposite on the two sides of the body. Magnets placed in this manner anywhere else on the two sides of the body do not influence muscle strength, as observed by manual muscle testing, unless they are placed over an active acupuncture point which responds to the magnetism. After balancing the muscles of the hyoid, magnets held lateral to it will no longer cause a previously strong indicator muscle to weaken.

These factors observed on a clinical basis show the need for much more extensive study of the neuromuscular suspension of the hyoid and its relation to equilibrium and organization between the two sides of the body. The need for considering this mechanism in the total organization of the stomatognathic system has been mentioned several times in the literature,^{5, 11, 21, 24} but there has been little electrophysiologic research done to understand the mechanism.

BODY LANGUAGE

There are several types of body language which indicate that hyoid neuromuscular balance should be evaluated. The most frequent indicator is dysfunction of any of the muscles of the closed kinematic chain of the stomatognathic system. This includes the cervical flexors and extensors, muscles of mastication, and orofacial muscles. It will often be seen that there is a reactive muscle²⁶ relation between muscles of the hyoid and other areas in the chain. It is frequently necessary to correct the hyoid muscle(s) to gain permanent correction of a remote muscle, because inappropriate stimulation to the muscle propriocep-

tor in the hyoid muscle causes poor function in a remote muscle. This is often observed when phonation, deglutition, tongue movement, or change of head position causes the examination of a remote area to become positive when it was previously negative.

Disorganization of the two sides of the body may relate with disturbance in the hyoid neuromusculature. This is evidenced by switching, reaction to bilateral brain activity, continued requirements for cross crawl patterning, or problems in the gait mechanism.

Hyoid Neuromuscular Balance Examination and Treatment

As with many other examinations of the stomatognathic system, it is necessary to test the hyoid muscles indirectly since they cannot be evaluated with the usual manual muscle tests. The system of examination developed by Goodheart⁷ involves moving the hyoid in various directions to stretch the muscles. When the hyoid is moved in such a manner that a previously strong indicator muscle weakens, it is suspected that a neuromuscular spindle cell within one of the stretched muscles is dysfunctioning. This sends a volley of afferent impulses into the neuronal pools, disturbing the system's organization, and a

previously strong indicator muscle weakens. An analysis of the muscles stretched by the hyoid movement indicates which muscle may be involved. The precise area of involvement is found by therapy localizing along the stretched muscles until the therapy localization is positive. Digital therapy is then applied, with the two points of manipulation coming together over the area. This is the neuromuscular spindle cell treatment to weaken a hypertonic muscle. This working hypothesis has been the one generally accepted in applied kinesiology. It is also possible that the manipulation influences muscle and

Hyoid Muscles and Function

fascia harmony, as in the fascial release technique in Rolf's work.²³ Treatment may also be applied to a trigger point as in Travell's²⁵ treatment. It has been questioned whether some of the muscles of the hyoid group contain neuromuscular spindle cells.^{6, 13, 19} Some of the questions which arise from the therapeutic efforts and clinical success of manipulating the hyoid muscle complex must await more thorough electrophysiologic evaluation of this complex mechanism.

Therapy localization to find the apparent neuromuscular spindle cell dysfunction should be done with close accuracy, such as using the patient's fingertip. Therapy localization to the hyoid bone is generally negative. It is only positive when, by sheer accident, the involvement of the muscle is close to its insertion and the muscle is therapy localized instead of the hyoid bone. There is also the possibility of positive therapy localization at the hyoid area from some other health problem; however, this is unusual. Makeup and other cosmetic creams may cause poor ability to therapy localize in this area.

The hyoid is challenged by having the patient hold the hyoid bone between his thumb and forefinger and move it into various positions directed by the physician. When a hypertonic muscle is stretched, a strong indicator muscle weakens. Prior to challenging, the patient should touch the hyoid and a previously strong indicator muscle should be tested to make certain there is no positive therapy localization without motion. If there is, evaluate to determine the type of dysfunction causing the positive finding. The hyoid challenge is done by holding the hyoid right and left, superiorly and inferiorly, anteriorly and posteriorly, and with various directions of torque. The physician should have the anatomy of the hyoid muscles well in mind so that he can determine which muscles are being stretched in the various hyoid positions.

A quicker method of challenging is to have a support person contact the hyoid and move it into the various test positions. This reduces the need for the doctor to describe the positions of hyoid movement; usually, more accurate movement can be accomplished rapidly.

The quickest method for determining the muscle most likely involved combines several challenges. For example, challenging the hyoid anteriorly stretches the posterior belly of the digastric and stylohyoid muscles. If a strong indicator muscle weakens, these muscles are probably involved. The hyoid can then be moved anteriorly on one side only and then the other, to determine which side should be further evaluated by therapy localization along the muscle. The illustrations show the various types and

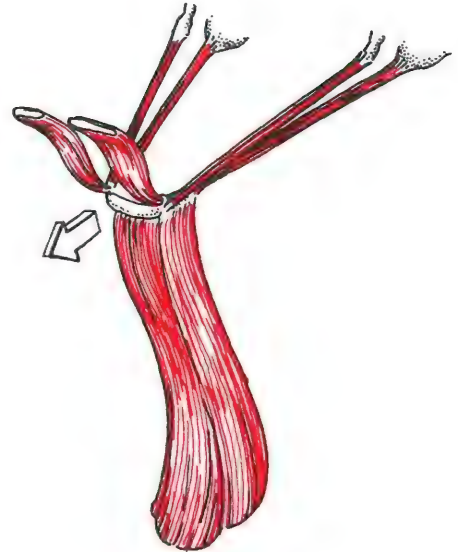
combinations of challenge, along with the muscles most likely involved.

Challenge as indicated in the illustrations will most often identify the muscle(s) needing treatment. In some cases it is necessary to use a combined hyoid challenge to locate the involvement. The types of combined hyoid challenge are anteroinferior, posteroinferior, anterosuperior, posterosuperior, and opposite rotation in the transverse and frontal planes. Opposite rotation in the transverse plane would be pushing anteriorly on the hyoid on one side and posteriorly on the other.

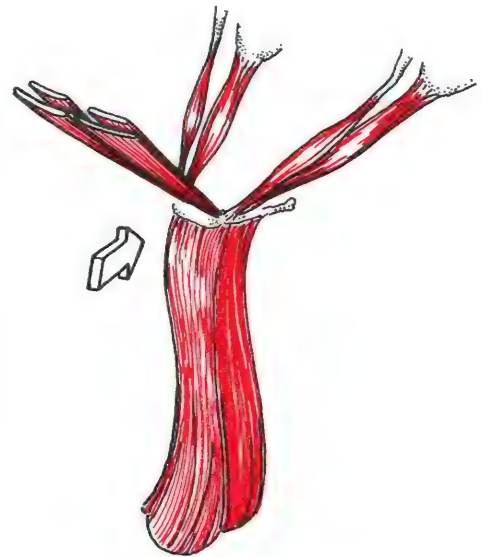
When a strong indicator muscle weakens on challenge, have the patient therapy localize along the muscles that are stretched when the hyoid is moved in the direction causing positive challenge. When a previously strong indicator muscle weakens from the therapy localization, there is good indication that the area of the muscle needing treatment is located under the therapy localizing finger. Neuromuscular spindle cell treatment is used to weaken the muscle. This is accomplished by the physician placing his thumbs directly over the point of positive therapy localization and pressing them together in alignment with the muscle fibers. It may be necessary to repeat this maneuver three or four times before positive therapy localization is eliminated. It generally requires approximately four to six pounds of pressure to gain an adequate response. The treatment is usually more comfortable for the patient if lotion is applied to the skin so the treating fingers slide easily.

Sometimes neuromuscular dysfunction of hyoid balance will not be apparent on challenge as described above. This appears to be the result of the dysfunction operating on a reactive muscle basis with the activities of swallowing and phonation. In problem cases of the stomatognathic system, or other instances of apparent hyoid neuromuscular involvement where the challenge is negative, have the patient add swallowing and phonation and jaw opening and closing while the challenge is done. When a positive challenge is found under these circumstances, final evaluation of the muscle involved and the location of muscle dysfunction are obtained with therapy localization as described above. Treatment is also provided in a similar manner.

Because the stomatognathic system is involved with total body function, there will be times when dysfunction of the neuromuscular balance of the hyoid does not reveal itself until the patient is in a weight-bearing position, either sitting or standing. In this case the hyoid imbalance revealed by challenge should be treated as usual; in addition, the weight-bearing mechanisms of the spine, pelvis, feet, etc., should be evaluated.

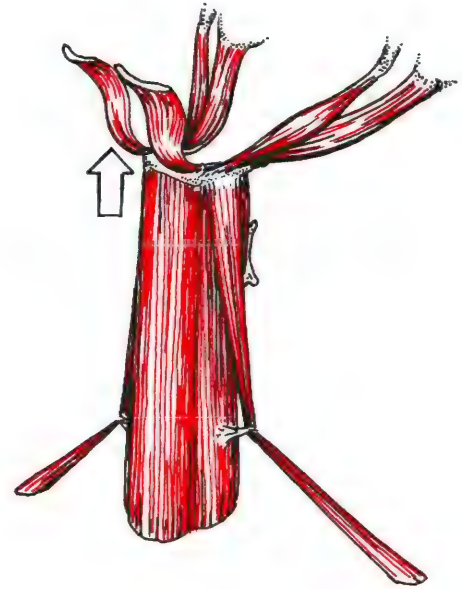


13—13. Anterior challenge stretches the posterior belly of the digastric and the stylohyoid muscles.



13—14. Posterior challenge stretches the anterior belly of the digastric, geniohyoid, and somewhat the mylohyoid muscles.

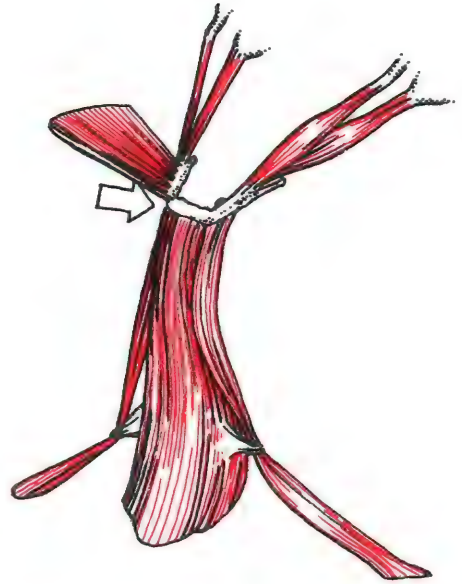
Hyoid Muscles and Function



13—15. Superior challenge stretches the omohyoid, thyrohyoid, sternohyoid, and perhaps the sternothyroid muscles.



13—16. Inferior challenge stretches the mylohyoid, geniohyoid, both bellies of the digastric, and the stylohyoid muscles.



13—17. Lateral challenge stretches the posterior belly of the digastric, stylohyoid, mylohyoid, omohyoid, and perhaps the other hyoid muscles on that side.



13—18. Various combinations of hyoid challenge can be done to help isolate the muscle(s) involved.

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Chapter 14

AK Examination and Treatment of the TMJ and Muscles of Mastication

Introduction

Throughout this text there has been an emphasis on the interactions occurring within the stomatognathic system, and that system's interaction with the rest of the body. As we proceed with the applied kinesiology examination and treatment of the temporomandibular joint and muscles of mastication, the thought of integration with other function must be kept foremost in mind. Although this chapter will emphasize the localized examination, remember previous subjects such as the cranial primary respiratory mechanism, occlusion, and the neuromuscular balance of the hyoid bone. A summary of the integration of these subjects is presented in Chapter 16, and stomatognathic organization with total body function is presented in Chapter 17.

There has been an increased awareness of temporomandibular joint dysfunction, especially in the last decade. It seems only right that this joint and its associated musculature should receive more attention, because it is the most used joint in the body. It opens and closes approximately 1,500-2,000 times a day during its various motions of chewing, talking, thinking, loving, yawning, and snoring.^{1, 28} Add to this the masticatory muscles' activity in swallowing 1,500-2,000 times every twenty-four

hours^{3, 23, 45} and it becomes obvious that this is a structural complex which is continually active throughout the day and night, offering no relief for structural disharmony which may be causing localized or remote symptomatic patterns.

This chapter first covers body language and other factors which indicate that the temporomandibular joint and its muscles should be examined. A discussion follows on conditions which must be differentially diagnosed from conditions of the TMJ and its muscles. Many of the therapeutic approaches used in applied kinesiology to treat the TMJ complex are discussed in Volume I of this series. There is a brief discussion of the muscle proprioceptors, reactive muscles, and spray and stretch and fascial release techniques as they specifically apply to the temporomandibular joint complex. This discussion assumes that the reader has studied the basic subjects in Volume I and is familiar with applied kinesiology use of them. Finally, there is a discussion of the applied kinesiology examination of temporomandibular joint movement, how to differentially diagnose various muscular involvements, and what AK treatment methods are applicable.

AK Examination and Treatment of the TMJ and Muscles of Mastication

Examination

In certain types of temporomandibular joint dysfunction the symptoms are obvious. There may be severe pain, limitation of motion, or popping and cracking; obviously under those circumstances the area should be examined. On the other hand, dysfunction in this structure may cause severe health problems in a remote area of the body, giving no indication for TMJ examination unless the physician is aware of the many ways in which this structure can cause remote problems. Unfortunately it is quite common to ignore the TMJ and its muscles unless there are obvious symptoms relating to it. Farrar and McCarty¹² point out, "In attempting to determine if a patient has a TMJ problem we should not ask the patient, 'does your jaw joint hurt', . . ." They further point out that objective evaluation of the mechanism should be done regardless of the symptomatic pattern; much dysfunction is present in the absence of localized symptoms.

Since temporomandibular joint function is inter-related with so many remote problems and is so frequently involved on a functional basis, it is realistic to at least do a screening examination of this mechanism on every new patient. This type of examination, described later in this chapter, requires little time and can be very effective in finding a majority of problems in this area. A considerable amount of evidence indicating the need for a more thorough evaluation becomes available during history-taking and consultation. This information comes from what the patient tells the physician and from body language observed during the discussion.

Obvious body language relating to the TMJ is pain in the muscles of mastication, joint pain, and popping and clicking of the joint. These are observed during the stages of history-taking, consultation, observation, and palpation.^{1, 9, 13, 28, 29, 54}

HISTORY AND CONSULTATION

Whenever there is evidence of need to examine the cranial-sacral primary respiratory mechanism, there is also evidence to evaluate the activity of the temporomandibular joint. Because the activity of these two divisions of the stomatognathic system cannot be separated, it will be necessary during examination to determine which area is primary, or if involvement of both has developed simultaneously. If one is primary, initial treatment directed to it will usually be the most efficient method of obtaining a stable correction. Sometimes correction can be obtained only when the major therapeutic effort is directed to the primary condition. The case history often gives clues to help delineate the course of events which has led to the current status, and finally leads the physician to the primary etiology. Examples of primary and secondary conditions will be of value in helping understand the importance of this determination.

In the initial description of her complaint, Mrs. Daberkow relates that she has suboccipital headaches which are almost always present and sometimes extremely severe. She frequently has visual and digestive disturbances. On questioning by the doctor she states that these health problems do not necessarily coincide with her headaches, and that the visual problem occasionally includes double vision. She can usually bring the diplopia into single vision by holding her head in a certain position. Her

physician asks her to demonstrate this activity and observes masseter muscle contraction when she assumes the head posture that relieves the diplopia. Further questioning by the physician seems to rule out a weight-bearing problem since she does not gain relief when lying down, is not worse at the end of the day, and often has the problem upon awakening in the morning.

When the physician questions Mrs. Daberkow about a history of accidents, it is revealed that these symptoms developed soon after an auto accident; she was riding in the front passenger seat. The auto she was in rear-ended another which was stopped at a light. She was thrown forward; her knees were lacerated on the dashboard and her head hit the upper portion of the dashboard and windshield. The only injury related to the accident was to her knees, which were examined and treated by her general practitioner on the day of the accident. They have since healed satisfactorily and there seems to be no residual problem. She does not relate her headaches to the accident.

Mrs. Daberkow's consultation and history reveal some additional symptoms relating with the cranial nerves and cervical spine dysfunction. On specific questioning she states, "My jaw doesn't feel right and I have a lot of tension in this area." (She indicates the temporalis and masseter muscles on one side.)

The type of treatment Mrs. Daberkow receives is

AK Examination and Treatment of the TMJ and Muscles of Mastication

often dictated by the type of physician she consults. The case history and consultation indicate disturbance in the cervical spine, cranial primary respiratory mechanism, and temporomandibular joint and its muscles. Three different physicians may develop totally different case plans and obtain results. Before looking at how various physicians might approach this condition, let's see what has happened in this hypothetical condition.

The etiology is trauma from the accident where Mrs. Daberkow's head hit the dashboard and windshield, creating cranial faults. Mrs. Daberkow does not relate her symptoms specifically to the accident since the headaches didn't begin until about three weeks later. This delayed reaction is not uncommon with cranial faults. The reason for the delay is speculative; it may be that the cranial nerves are capable of resisting the early stages of peripheral entrapment from cranial faults. Symptoms may not develop unless the stomatognathic system dysfunctions further. This includes the closed kinematic chain, represented in this case by the masticatory muscles and the neck flexors.

Thorough evaluation by a physician knowledgeable in the total stomatognathic system reveals that Mrs. Daberkow has several cranial faults, bilaterally weak sternocleidomastoid muscles, imbalance of the muscles of mastication, hypertonicity of the cervical extensors, and hyoid muscle imbalance. The suboccipital headaches are secondary to the weak sternocleidomastoid muscles, which are causing the unopposed suboccipital muscles to become hypertonic almost to the point of spasm. Muscle hypertonicity reduces the blood supply to the muscles, causing pain; it may also impinge upon the greater and lesser occipital nerves with resulting neuralgia. Because of the muscular imbalances present, cervical subluxations and/or fixations have developed, causing additional symptoms. Cranial faults, in addition to creating the sternocleidomastoid weakness, have caused the masticatory and hyoid muscles to be out of balance, resulting in malocclusion and symptoms in the masseter and temporalis muscles. The cranial faults have also caused imbalance in the extraocular muscles, resulting in diplopia. Since Mrs. Daberkow has additional visual disturbances there may be distortion of the bony orbit, causing pressure on the eye or peripheral entrapment of cranial nerve II. Cranial faults are probably responsible for digestive disturbances, which could develop as a result of disturbance to the vagus nerve (cranial X). Now let's look at how different physicians might evaluate and treat this condition.

Case plan one is developed by a chiropractor who quickly observes the cervical subluxations

and/or fixations and proceeds to treat the condition with cervical manipulation. The case plan may also include electrotherapy to the hypertonic suboccipital muscles and other support to the cervical spine. This approach breaks into the vicious circle of events causing the symptomatic pattern from which Mrs. Daberkow is seeking relief. The sternocleidomastoid and upper trapezius, as well as the intrinsic muscles of the upper cervical area, can be disturbed by vertebral subluxations and fixations. Improved balance of these muscles which pull into the cranium may relieve enough stress on the cranial primary respiratory mechanism to allow the body to correct itself in its own self-correcting, self-maintaining way. With this assist from the body's own natural abilities, this case plan proves successful in correcting the condition.

Hopefully, this is the course of events. The successful illustration given depends on the sternocleidomastoid and trapezius imbalance being due to vertebral subluxations and fixations which are somehow secondary to imbalance of the closed kinematic chain of the stomatognathic system. This case plan, on the other hand, may actually create additional problems. If the weakness of the sternocleidomastoid muscles was primarily due to cranial faults entrapping cranial nerve XI at the jugular foramen, there would be no improvement in their function. Manipulation of the upper cervical region in this case could correct the condition only by accidentally placing strain on the dura mater attached to the 2nd and 3rd cervical vertebrae, which would luckily pull into the cranial primary respiratory mechanism to unlock the cranial faults. More likely, the manipulation to the upper cervical area would put additional stress into the already compromised structure.

Case plan two is developed by a dentist Mrs. Daberkow consulted because of the constant tension in her jaw. Malocclusion is quickly observed on examination and related to the imbalance in the closed kinematic chain of the stomatognathic system. The case plan is to first reduce stimulation to the periodontal ligament receptors by providing a bite plane. Soon after the appliance is installed and equilibrated, Mrs. Daberkow feels relief of the jaw tension. The dentist continues to equilibrate the bite plane as the muscles of mastication relax and function in a more balanced manner. With their improved balance they direct force into the cranium, activating the mechanism; again, in the body's own self-correcting, self-maintaining manner, they unlock the cranial primary respiratory mechanism and release peripheral entrapment on the cranial nerves. With return of balanced function the bite plane is removed; the dentist finds minimal malocclusion,

AK Examination and Treatment of the TMJ and Muscles of Mastication

unless he attempts to equilibrate to centric relation in the most retruded superior condylar position. Fortunately, the case plan included the initial use of a bite plane. If the case plan had directed initial effort toward equilibration, Mrs. Daberkow would probably have gotten no relief; worse, the cranial faults would have been locked in because the more stable mandible was made to fit the imbalanced but flexible maxillae.

The third case plan is developed by a physician knowledgeable in the cranial primary respiratory mechanism. The first clue that the problem is in the cranium is that most of Mrs. Daberkow's symptoms relate to cranial nerve dysfunction. The second clue is that Mrs. Daberkow can relieve her diplopia by tilting her head to a specific position. This is emphasized as the physician observes masseter muscle contraction in the position in which Mrs. Daberkow gains relief. Contraction of the jaw closer muscles brings the lower dental arch into intercuspation with the upper one. Since Mrs. Daberkow had a good occlusion prior to the accident, the solid mandible forces the distorted maxillae into an improved position, causing the diplopia to return to single vision. The etiology of trauma to the head is the third clue that the primary problem is in cranial function. If the physician can evaluate the cervical spine, cranial primary respiratory mechanism, and the occlusion, he can probably quickly confirm the cranial mechanism as primary. His examination of the three areas reveals cranial faults, muscle imbalance associated with the malocclusion (described later in this chapter), and upper cervical subluxations (described in Volume I).

Since case plan three is developed by a physician knowledgeable in all three areas, he can examine them simultaneously. In this examination, an effort is made to determine if challenge to the cervical spine will affect the TMJ and cranium, or whether the TMJ and its muscle balance will affect the cervical spine and cranium, or whether the cranium will affect the cervical spine and TMJ. (This type of integrated examination is explained more thoroughly in Section III of this text.) In this case it is found that the cranium is primary to the temporomandibular joint and cervical spine dysfunction. The TMJ and its muscle balance are examined with various phases of respiration typical in applied kinesiology evaluation of the cranial primary respiratory mechanism. When the patient holds a particular phase of respiration, the positive TMJ tests become negative. When the patient holds the same phase of respiration, challenge to the cervical vertebrae also turns negative. This is almost 100% indication that the cranial faults are primary. The physician finishes his examination of

the cranium and makes corrections, then re-examines the temporomandibular joint and cervical spine; they now test negative with no therapy applied. This example illustrates how important it is to find the primary condition. Good evidence that the primary problem has been found is when its correction simultaneously eliminates many other positive findings. Excellent feedback to the third physician is the spontaneous correction of the temporomandibular joint and cervical spine following the cranial correction.

An example of a different etiology and primary condition is supplied in the case of Mr. Stovall. During consultation, he describes chronic sinus trouble which causes severe pain. He points to the maxillary area and describes the pain as being so severe that it sometimes feels like there is a hot poker behind his eye. Occasionally the pain is in the frontal area, but more often it is in the maxillary area. This problem has gotten progressively worse over the past five years. He has sought the services of several physicians, and has tried some home remedies involving diet change and nutrition. Over the years he has used numerous decongestants and analgesics, received antibiotic treatment, and had surgical drainage on two occasions. He has eliminated dairy products from his diet, gone on a low carbohydrate diet, and used voluminous amounts of vitamins C and A. He believes he may be allergic to some proteins since he develops digestive gas upon their ingestion.

When the physician asks questions about other types of health problems, little additional information is obtained. Specific questions relating to weight bearing, such as fatigue at the end of the day, foot or low back problems, worsening of symptoms after standing for prolonged periods, or relief upon lying down, reveal only that leg cramps may develop if he is on his feet for a prolonged time. Mr. Stovall strikes the physician as one who complains little about his health and tends to overlook problems about which others would complain. Specific questions regarding his dental health are answered with, "Oh, my teeth are in excellent condition. I spent \$1,800 on them and the dental hygienist cleans them once a year." Additional questions reveal that he had four crowns placed five years previously. He notes that he is having some discomfort in his back teeth, but it is getting close to time to go to the dentist anyway. Observation reveals periodontal problems developing in the lower posterior quadrants.

Mr. Stovall's consultation and history again show dysfunction of the cranial-sacral primary respiratory system to be the cause of his sinus disturbance, and two ways in which the cranial faults could be

AK Examination and Treatment of the TMJ and Muscles of Mastication

responsible for the sinusitis. The first is direct disturbance to the sinuses which often results from cranial dysfunction. Misalignment of the cranial bones can disturb sinus drainage and interfere with the nerve supply to the sinuses. The second is shown in Mr. Stovall's body language which indicates probable hypochlorhydria, relating with the digestive gas after eating protein foods and possibly with the leg cramps as a result of calcium deficiency. Protein digestion and calcium absorption both depend upon an adequate amount of hydrochloric acid. In applied kinesiology, hypochlorhydria and calcium deficiency are related with allergic reactions (see Volume V) which could be the cause of Mr. Stovall's sinus problems.

To understand how Mr. Stovall will respond to different therapeutic approaches, it is necessary at this time to know how his problem developed. During his extensive dental procedures five years previously, two third molars were extracted and four crowns were placed as reconstruction procedures. The crowns were poorly equilibrated and encroached on the freeway space, changing the vertical dimension. The increase in vertical dimension may have been done as a result of the dentist's philosophy, which indicated a greater vertical dimension was necessary, or it may simply have been the result of a poor case plan. Because of the resulting malocclusion, the muscles of mastication pulled on the cranium in an imbalanced manner, creating the cranial faults responsible for Mr. Stovall's symptoms. The repeated trauma of the malocclusion has now resulted in periodontal disease, evidenced in the lower posterior quadrants. Again, let's observe three different case plans developed by different doctors and the resultant effects on Mr. Stovall's condition.

Case plan one is developed by a chiropractor to whom Mr. Stovall was referred because one of his friends obtained good results for a sinus condition. Examination reveals imbalance in the muscles of the cervical spine; motion palpation of the vertebrae reveals several areas of aberrant movement. This is very common when any area of the stomatognathic system is involved because the closed kinematic chain becomes imbalanced. Shortness of a leg is observed when the patient is supine, the pelvis appears imbalanced, and there are subluxations of the lumbar vertebrae. The chiropractor's case plan includes a heel lift for the apparent short leg and spinal manipulation of the areas of vertebral subluxation indicated by motion palpation and x-ray misalignment. He also includes hydrochloric acid supplementation for the muscle cramping and for protein digestion. The results of this treatment depend on luck. It is possible (though not probable) that

manipulation in the cervical and lumbar spine, and change of general spinal and pelvic balance as a result of the heel lift, will structurally change the balance within the stomatognathic system sufficiently to change the cranial faults, or perhaps even clear them. If the cranial faults are simply changed to a different type the sinus condition may improve, but new and probably unrelated symptoms may eventually develop from the different peripheral nerve entrapment resulting from new cranial faults. It is fortunate for the doctor that he will receive credit for correcting the sinus condition and won't be blamed for new symptoms unless they seem to be specifically related, such as low back pain, etc.

The second case plan is developed by a physician knowledgeable in the cranial-sacral primary respiratory mechanism but unaware of its close relation with the TMJ, occlusion, and muscles of mastication. Treatment is directed to the cranial faults and the doctor's examination, whether by palpation or muscle testing, indicates improved cranial function. Unfortunately, when the patient returns for subsequent visits the cranial faults have also returned. With the continuing cranial manipulation there may be sufficient change in the mechanism to bring at least temporary relief from the sinus condition, or the cranial faults may be shifted to a different type and relieve the sinus condition completely but bring new symptoms, as happened in case plan one. Here again, the doctor may receive credit for improving the sinus condition and not have the new symptoms related to his treatment.

The third case plan is developed by a dentist Mr. Stovall is consulting for the first time. The change of dentists is the result of Mr. Stovall having a job transfer to a different city. The very obvious periodontal disease causes the dentist to evaluate for traumatic malocclusion, which is quickly apparent. The case plan includes a mandibular bite plane constructed to splint the lower teeth. It stabilizes the teeth and eliminates stimulation to the periodontal ligament proprioceptors, allowing the adaptive program to be reduced. The crowns are subsequently equilibrated for proper freeway space and occlusion, and Mr. Stovall's periodontal problem subsides. Because of the balanced occlusion and normal vertical dimension, the cranial primary respiratory mechanism is no longer under stress; the cranial faults self-correct and the sinus condition subsides.

There is an irony to this case plan and the results of the third hypothetical situation. Mr. Stovall obviously will be very grateful to the dentist for the improvement in the periodontal problem; however, neither Mr. Stovall nor the dentist may ever understand why the sinus condition "spontaneously"

AK Examination and Treatment of the TMJ and Muscles of Mastication

subsided after five years. The dentist may not even be aware that Mr. Stovall had a sinus condition.

These two hypothetical patients are representative of the interplay taking place within the stomatognathic system and how important it is to find the primary condition rather than treat the symptoms. In some instances the examining doctor is the appropriate one to manage the patient's total treatment; in other cases it may be necessary to refer the patient to a practitioner of a different discipline for optimum results. In many chronic conditions it is necessary for the patient to be treated by two or more physicians in different disciplines to obtain the optimum correction. The combined types of examination available in applied kinesiology help delineate the primary cause of the health problem.

Both of these hypothetical cases are examples of involvement of the total stomatognathic system with only one area being primary. The trilogy could be completed by creating a hypothetical case which has trauma to the cervical spine, such as a whiplash injury, as the initiating factor. Three physicians could develop case plans for correction. Two of them might or might not relieve the condition by disrupting the vicious circle of events, and one could have the right approach of correcting dysfunction in the cervical spine.

This interaction of the stomatognathic system demands that a physician be aware of the body language of dysfunction in all areas of the system, and how those areas interrelate with each other. Whenever there is body language of cranial dysfunction (Chapters 2 and 6), hyoid muscle imbalance (Chapter 13), or cervical spine dysfunction (Volumes I and IV), the TMJ, muscles of mastication, and the occlusion should be thoroughly evaluated. They will frequently be involved, either on a primary or a secondary basis.

OBSERVATION

With the patient seated or standing, have him simply open his mouth. The mandible should drop straight down with no special effort on the patient's part. Muscular imbalance or disc dysfunction is evidenced by the mandible swinging to one side and back to the center, or staying off-balance. The most common muscular cause of mandibular deviation on opening is an imbalance of the external pterygoid muscles. Other muscles, especially the posterior fibers of the temporalis, can also be involved. Another cause of lateral mandibular deviation is poor harmony of the TMJ disc with joint function. Usually there is an anterior disc on one side, causing the

condyle not to move smoothly. Popping, clicking, or crepitation is usually present with this condition.

Often a patient will tilt his head back as his mouth opens. Goodheart calls this "opening the head instead of opening the jaw." The most efficient method of opening the mouth is simply dropping the mandible. When the head tilts back, as in "opening the head," there is generally imbalance in the stomatognathic system or poor general use of the body's postural muscles; this is a clue to examine for these conditions.

While in the observation phase, the dentition should be evaluated. Naturally this stage of examination is overlooked only by the non-dental physician who may possibly feel that's not his province. Without this evaluation, examination of the stomatognathic system is incomplete. It is certainly not necessary to do a thorough dental evaluation, but the general nature of the occlusion, signs of orofacial muscle imbalance (Chapters 4 and 15), the approximate amount of freeway space (Chapters 11 and 12), and periodontal health should be observed, as well as the status of dental repair, prophylaxis, and any missing teeth.

The amount of mandibular opening should be noted. It is generally considered that the proximal interphalangeal knuckles of the first, second, and third fingers on the non-dominant hand should fit between the upper and lower incisor teeth when the mouth is actively opened to its maximum amount.^{6, 10} If the range of motion is limited, it should be recorded for future reference. Although this measurement is valuable in relating with future therapy, it is not pathognomonic of temporomandibular joint dysfunction. Many patients with TMJ dysfunction can open the jaw to meet the three-knuckle rule; conversely, some patients having no problem with the TMJ or muscles of mastication cannot open the jaw to meet the rule. A study by Rieder⁴⁷ revealed no statistically significant correlation between limitation of jaw opening and temporomandibular joint dysfunction.

PALPATION

The use of palpation early in the examination can save considerable time, as well as provide additional information about the condition. Included in this phase of examination are the joint itself and the muscles of mastication.

Joint Palpation

Often this phase of observation will clearly indicate aberrations in joint movement. There may be an audible pop on opening or closing, and a

AK Examination and Treatment of the TMJ and Muscles of Mastication

visually observed rapid shift in condylar position.

Most palpation information about temporomandibular joint function can be obtained by placing fingers in each external auditory meatus, with the finger's fleshy pad toward the condyle. Unless the physician has relatively small fingers it is usually best to use the little finger while facing the supine, sitting, or standing patient. Crepitation, popping, and clicking can easily be palpated, along with general smoothness of the condyle's movement from rotation to translation. As the mandible opens the motion should be smooth and bilaterally symmetrical. When there is chronic joint strain or pathology there will usually be pain when mild pressure is exerted toward the articulation through the external auditory meatus. A stethoscopic examination of the joint will also reveal low-level sounds of clicking or popping.

Temporomandibular joint motion can also be palpated at the lateral aspect of the joint. It is sometimes easier to observe bilateral asymmetry from this position, especially if the deviation occurs in the middle or latter part of the condyle's translation on the articular eminence. Examination here includes palpating for capsular swelling and other signs of joint disease, such as heat in rheumatoid arthritis and infection, morphology of neoplasms, etc.



14—1. While palpating the TMJ from within the external auditory meatus, have the patient move the mandible through its range of motion and observe for crepitation or popping.

This lateral location is where the patient will place his fingertips for future therapy localization examination. It is valuable at this point to show the patient where the temporomandibular joint is. Have him feel the clicking that takes place if there is crepitation,

popping, or cracking. Say to the patient, "Mr. Stovall, feel right here for the clicking in your jaw. Note the location of your jaw joint just in front of the little bump (tragus) of your ear. I'm going to have you place your fingers there later in the examination." When the physician asks patients to place their fingers on the jaw joint, there is a tendency for them to be too low. Making a specific note of the joint's location at this stage saves time later in the examination.

The joint can be further evaluated by observing passive range of joint motion. The examiner grasps the anterior portion of the mandible with his thumb over the lower incisor teeth and the index finger wrapping around the lower anterior border.

Many cases of chronic TMJ strain and those with pathology will have a posterior capsulitis. Pain is increased in this localized area as the examiner gently moves the mandible posteriorly to place strain between the condyle and the posterior fossa. If there is no discomfort with the gentle posterior movement, a little more forceful evaluation can be done.

The range of TMJ motion previously observed on an active basis can be further evaluated passively by the examiner manipulating the mandible with the anterior contact mentioned above. When motion is limited, the examiner should not force the jaw but should evaluate for pain created during the passive movements. This information can be used for comparative purposes after treatment to the TMJ.

Palpation of Muscles of Mastication

The muscles of mastication are palpated for hypertonicity, pain, trigger points, and fibrous masses which probably relate to dysfunctioning Golgi tendon organs or neuromuscular spindle cells. The "jump sign"⁵⁵ of Travell's⁶¹ trigger point technique and the palpatory indications for various types of treatment are discussed later in this chapter with the applied kinesiology examination and treatment of the TMJ and muscles of mastication.

Temporalis. The broad fan-like temporalis muscle should be palpated first with a light digital contact and then with a deeper one because of the heavy investing temporal fascia covering the muscle. Care must be taken not to confuse active points on the temporal sphenoidal line⁶⁶ with palpatory findings of the muscle itself. The three divisions of the temporalis should be considered individually, as they refer pain differently from trigger points.⁶¹

The posterior fibers, which are almost on a horizontal plane above and posterior to the ear, are more often found to be involved in applied kinesiology diagnosis. Trigger points in this area refer pain to the upper molar teeth and to the occiput. These

AK Examination and Treatment of the TMJ and Muscles of Mastication

fibers are often hypertonic, requiring neuromuscular spindle cell or fascial release technique. Hypertonic posterior fibers are often present when an individual has had the occlusion equilibrated to match a retruded superior centric relation condylar position. They are also frequently present when there is an anterior disc with an opening click or lock.

Trigger points in the middle fibers refer pain to the upper dental arch from approximately the canine to the second pre-molar teeth and to the temple, and occasionally to the temporomandibular joint. Trigger points in the anterior portion of the temporalis refer pain to the supraorbital ridge and into the upper incisor teeth.

General hypertonicity of the temporalis muscle is frequently responsible for temporal headaches. There is often a jamming of the squamosal suture which may require strong, sustained separation techniques.

Masseter. If the masseter is found hypertrophied on palpation, bracing or bruxism should be considered. The most common abnormal finding in the masseter muscle is the nodular, ropy, or puffy swelling indicative of a malfunctioning neuromuscular spindle cell.

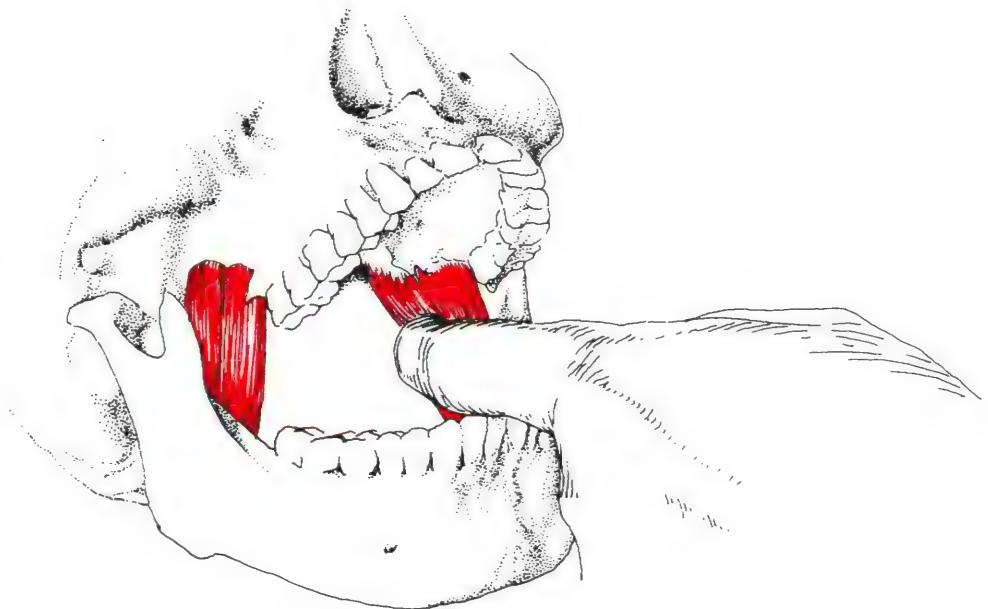
Trigger points in the superficial layer refer pain to the general area of the jaws. The upper portion of this division of the muscle refers pain to the molar teeth and gingiva of the upper arch, while the lower part causes pain in the lower molar teeth and gingiva. Trigger points in the insertion area at the angle of the mandible refer pain to the outer end of the eyebrow. Trigger points in the deep layer of the masseter refer pain to the TMJ and deep into the ear.

Internal Pterygoid. The internal pterygoid is palpated with the patient holding his jaws wide open. Palpation usually begins at the muscle's insertion on the ramus of the mandible and progresses superiorly to the origin on the lateral pterygoid plate and pyramidal process of the palatine bone. There will often be considerable tenderness in this muscle, along with the nodular, ropy, swollen feeling of neuromuscular spindle cell dysfunction, but it requires considerable experience to

feel it. Dawson⁹ considers tenderness in any of the pterygoid muscles as indicative of malocclusion.

Trigger points located in the internal pterygoid muscle refer pain to the tongue, posterior hard palate, and TMJ. Pain is not noted to be referred to the teeth from this muscle.

External Pterygoid. The most difficult masticatory muscle to palpate is the external pterygoid. In cases of temporomandibular joint dysfunction it is often very tender. Routine palpation for diagnostic information is usually not done; rather, when there is evidence the muscle must be treated, palpation for diagnosis and pressure for therapy are done simultaneously. Only portions of the external pterygoid can be digitally contacted by the physician. The varying anatomical arrangements of individual patients sometimes allow a better contact from a lateral approach to the muscle and sometimes from a medial one. In both cases the patient holds his jaws open wide. To approach the muscle from its lateral aspect, the physician guides his index finger past the buccal surfaces of the upper molar teeth to the lateral pterygoid plate and continues to direct his finger along the inferior and as lateral as possible aspect of the external pterygoid muscle, continuing toward the neck and condyle of the mandible. The medial approach is to direct the index finger along the lingual surface of the molar teeth to the pterygoid process, and along the external pterygoid muscle with the finger approaching the neck and condyle of the mandible from the medial side. Both approaches



14—2. Palpation of internal pterygoid.

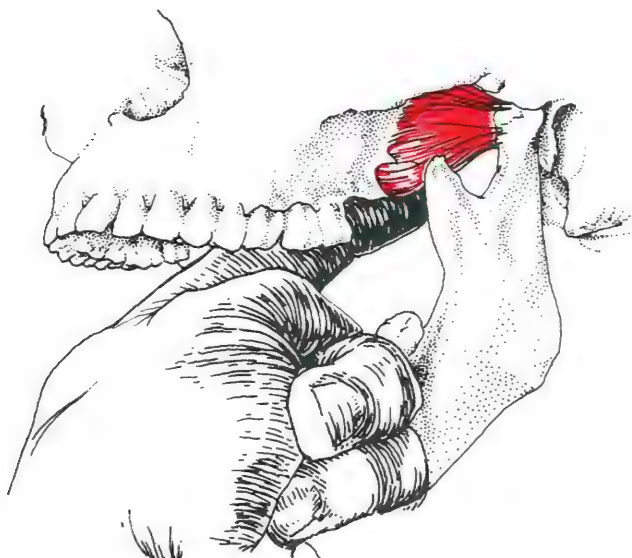
AK Examination and Treatment of the TMJ and Muscles of Mastication

require a gentle touch because there is often extreme tenderness of this muscle when there is temporomandibular joint dysfunction. Only with considerable experience will the physician be able to feel the nodular swellings of a suspected neuromuscular spindle cell dysfunction.

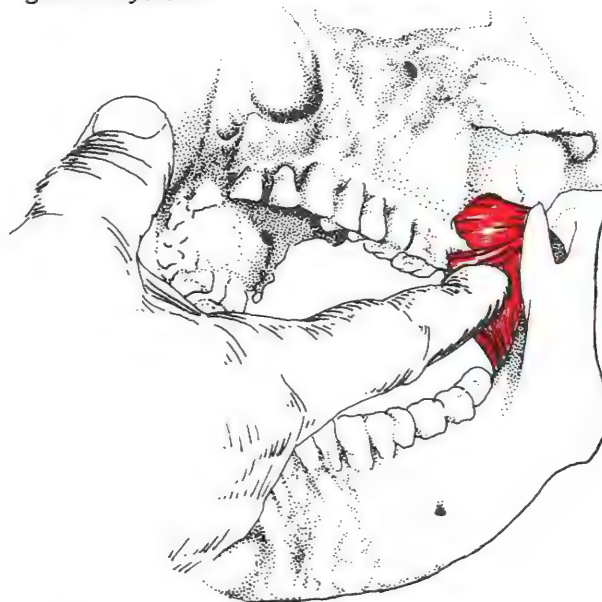
Trigger points in the external pterygoid refer pain within the temporomandibular and maxillary regions. Because of the muscle's inaccessibility, the patterns

of the two divisions have not been clearly separated.⁶¹

There are many other muscles which are palpated in the total examination of the stomatognathic system. Many of these have trigger points which refer pain into the masticatory apparatus. Important among these are the upper trapezius and sternocleidomastoid. Their examination is discussed in Chapter 16 with the total organization of the stomatognathic system.



14—3. Medial approach to external pterygoid.



14—4. Lateral approach to external pterygoid.

Differential Diagnosis

Many different conditions may refer pain into the stomatognathic area. Some of these are described pain pathways which are well-known, yet there is considerable pain referral into the area which the current data base of knowledge does not adequately explain. In addition to differentially diagnosing referred pain, it is necessary to delineate pathology in the area for which the conservative treatment approaches described in this text are not applicable. This summation of differential diagnosis should be combined with the physician's previous knowledge of differential diagnosis to supply the comprehensive knowledge necessary to evaluate this area. It is very important to recognize neuropathies, intracranial lesions, neoplasms, bone dyscrasias, and other conditions which may require additional therapeutic work-up by specialists in those areas.

There is probably no other area in the body which requires as much interprofessional communication as the stomatognathic system. The physician knowledgeable in standard differential diagnosis of the area is aided by the functional evaluation afforded

by applied kinesiology. During the diagnostic work-up which includes applied kinesiology, all aspects of the examination should provide additional support to previous information, culminating in an understanding of the condition which does not have conflicting information. Furthermore, therapeutic efforts should affect the body in predictable ways. When evaluations and functional treatment do not integrate for a predictable outcome, further investigation is indicated because the basic underlying cause and condition have probably not been determined. Some of the more common and unusual referred pain patterns and conditions are summarized here, but it should be recognized that the scope of this text does not permit a comprehensive discussion of differential diagnosis.

It is convenient to group differential diagnosis into intra- and extracapsular conditions. The intracapsular conditions are primarily of the joint itself, while the extracapsular — sometimes quite remote from the temporomandibular joint — refer pain simulating disturbance in the joint.

INTRACAPSULAR TMJ PATHOLOGY

There are two basic divisions in pathology of the temporomandibular joint: bone dyscrasias and disc dysfunction or pathology. The temporomandibular joint exhibits most of the pathology that other freely movable joints of the body do. Radiologic investigation and blood laboratory analysis are important parts of diagnosing intracapsular disorders.

Degenerative Joint Disease (Osteoarthritis)

The TMJ responds to traumatic insult with degenerative joint disease just as other joints of the body do. It may result from chronic repeated microtrauma of joint strain from imbalance of other components of the stomatognathic system. Extrinsic acute trauma of greater magnitude may be the etiologic factor of subsequent degenerative joint disease. Often the condition is slow to develop, but it can be more rapid when the joint experiences severe or constant trauma. The insidious type of degenerative joint disease is more often found in the elderly, as with the condition in other joints of the body. Often degenerative joint disease is secondary to strain in the joint from the loss of posterior teeth and shrinkage of the ridges underneath dentures.⁴⁶ Degenerative joint disease develops much more rapidly in an individual who has chronic relative protein deficiency and disturbance in mineral deposition, such as calcium. This often develops when digestive disturbances are present, such as hypochlorhydria.

There is generally pain in the region of the temporomandibular joint, and movement causes crepitation and sometimes cracking and popping. Initial evidence of the condition is recurrent discomfort which becomes more frequent and severe, with possible limited motion as the degeneration progresses. The osteoarthritic cycle of pain upon arising with symptoms improving as the day progresses only to re-appear toward the end of the day, is usually present. Often there is degenerative disease in other joints of the body. The presence of Heberden's nodes, which usually develop at the distal interphalangeal articulations, helps diagnose degenerative joint disease in temporomandibular joint dysfunction. Clinical findings should be combined with radiologic evaluation.

Radiologic findings are disc loss, bone production seen as spur formation, and changes in joint structure. Eburnation may develop diffusely in the condylar head and mandibular fossa. Joint space between the fossa and head may be completely lost from thinning and fraying of the disc, although even with advanced degenerative joint disease changes,

considerable joint motion is retained; fibrous and bony ankylosis rarely occurs.³⁸

Rheumatoid Arthritis

When rheumatoid arthritis is classically definite in the body, a large percentage of patients manifests it in the temporomandibular joint. A study of sixty-five patients revealed that 78% of the joints (86% of patients) had structural alteration from the rheumatoid condition; only 55% had reduction of mobility.¹¹ There are, however, studies that indicate a much lower percentage.²⁶

Rheumatoid arthritis is exacerbated by physical or emotional shock, fatigue, trauma, infections, and exposure to rain or cold. In applied kinesiology this is recognized as stress to the adrenal gland. (AK's approach to rheumatoid arthritis, as well as to degenerative joint disease, is discussed in Volume V of this series.) The patient should be evaluated for crepitus and joint tenderness. There is frequent limitation of motion, along with local swelling, heat, and pain in the joint area. Pain may radiate to the teeth and jaws, neck, and the ear, and it may be impossible to close the mouth.²⁷ When rheumatoid arthritis is found in the temporomandibular joint, it is frequently present in other joints of the body. The characteristic swelling, deformity, and heat at other joints help differentially diagnose rheumatoid arthritis from the Heberden's nodes characteristic of degenerative joint disease. Laboratory evidence of rheumatoid arthritis includes increased erythrocyte sedimentation rate and a positive latex fixation test.⁷

Radiographs may show demineralization, joint space narrowing, and bone erosion or production. These findings are similar to those in all rheumatoid arthropathies and some other joint diseases. Differential diagnosis by x-ray may be impossible.³⁸

Gouty Arthritis

The temporomandibular joint is not a common site for major involvement with gouty arthritis. When gout is present it increases the pain of degenerative joint disease in the TMJ.² Reducing the elevated uric acid level and balancing TMJ dysfunction will often relieve the pain.

Osteochondritis

(Idiopathic Condylar Osteonecrosis)

Ninety-five percent of osteochondritis cases occurs in females fourteen to seventeen years of age, hence the term "cheerleader's syndrome." This is not as common an area of ischemic bone necrosis as other joint areas. Radiographic findings of avascular

necrosis with possible fragmentation should be differentially diagnosed from psoriatic arthritis.

Psoriatic Arthritis

A primary distinguishing factor of psoriatic arthritis is the skin lesions of psoriasis, along with peripheral arthritis and spinal involvement of syndesmophytes observed on radiographs.⁶⁸ There is usually a moderately elevated erythrocyte sedimentation rate and white blood cell count. Uric acid will be normal, although there is some controversy about this.

Infectious Arthritis³⁴

Infectious arthritis is not common in the temporomandibular joint; however, it should be diagnosed early because of the damage the joint can suffer. Infectious arthritis is usually localized in one articulation and found in the larger joints of the body. It is possible for a complicating infection to be present in rheumatoid arthritis, degenerative joint disease, and other pre-existing joint disease. During the first week of the condition x-rays may be negative; however, they will soon demonstrate rapid destruction. By the time soft tissue swelling occurs, osteoporosis is usually present. This joint disease may result from local extension of infections in the middle ear, mastoid, or parotid gland.⁶⁴

Ankylosis of the TMJ

Ankylosis of the joint is secondary to rheumatoid arthritis, rheumatic fever, joint infection, fractures of the condyle, or prolonged immobilization of a severely damaged condylar head.⁶⁴

Congenital Malformations

Malformed condyles can give rise to symptoms simulating temporomandibular joint arthrosis.⁵⁴ There may be condylar hyper- or hypoplasia, either of which can cause malocclusion.¹²

Neoplasms

Osteomas may develop on the condylar head, causing limited motion. Demonstration on radiographs is the final diagnosis, but Vamvas⁶⁴ recommends evaluation by passive stretch. When motion is limited by a bony obstruction, there will be a sudden stop of mandibular movement when the obstruction is hit. When the passive pressure is removed, there will be no rebound as is present when muscle has been stretched, limiting the range of motion. Chondromas may grow on the condyle, coronoid process, articular eminence, or the zygoma, causing limitation of movement and pain. Some of these may need to be surgically removed.¹²

Luxation or Subluxation

Luxation (dislocation), discussed in Chapter 12, is easily recognized. Some consider subluxation to be a disarticulation of the disc with the condylar head. This has been referred to previously in this text as displacement of the disc. In any event, there is less misalignment than with a luxation. Subluxation is generally considered to be incoordination of the temporomandibular joints as a result of muscular imbalance.⁶ The patient often complains that his teeth do not mesh together correctly. It often develops as the result of occlusal treatment. Schwartz⁵² and Brod⁶ recommend that the occlusion not be equilibrated; in fact, the condition often develops as a result of occlusal equilibration. This is a condition which responds very well to the applied kinesiology evaluation and treatment described later in this chapter.

Trauma

Reaction of the temporomandibular joint to trauma must be considered in two categories: acute reaction and chronic manifestations. Acute trauma is usually the result of a blow to the mandible which causes a jamming force into the TMJ. It may cause a transitory problem or produce permanent damage to the joint structure. Acute trauma may also result from less physical acts, such as prolonged mouth-opening procedures in dentistry, mouth and throat surgery, bronchoscopy, etc.

Frequently the stomatognathic system, including the temporomandibular joint, develops severe problems from the hyperflexion-hyperextension cervical injury, often called a whiplash. Cranial faults are frequently a part of this complex injury, either on a primary or a secondary basis. Treatment applied by cervical traction may produce iatrogenic conditions in the cranial primary respiratory mechanism and the temporomandibular joint. When there is loss of vertical dimension — whether it be from loss of teeth, intrusion, or whatever — caution should be exercised in applying cervical traction. Applied kinesiology methods of treatment nearly always provide an approach superior to cervical traction.

Temporomandibular joint trauma of a chronic nature is from muscular imbalance, loss of vertical dimension, disparity between centric relation and centric occlusion, and other factors which create repeated microtrauma to the articulations. Often this type of involvement is insidious, eventually manifesting itself as jaw popping, grating, muscular spasm, jaw pain, etc.

EXTRACAPSULAR DISTURBANCE REFERRED TO THE TMJ

There are many conditions within and without the broad definition of the stomatognathic system which can simulate temporomandibular joint pathology. In most instances when writers refer to temporomandibular joint disturbance, they are speaking of the joint and the muscles of mastication. Our first consideration is to recognize that dysfunction of the masticatory muscles can cause referred pain to the joint. Imbalance in the joint from improper muscle action can cause direct stress in the joint. The major discussion here considers conditions which can refer pain to the temporomandibular joint and the muscles of mastication.

Teeth

Pathology of the teeth does not often refer pain directly to the temporomandibular joint. Their status should be considered whenever pain is thought to come from temporomandibular joint dysfunction. Possible pain radiation from the teeth ranges from the supra-orbital region throughout the facial area to below the mandible. Occasionally pain can even radiate to the back of the head.

Localizing dental pain is often done by percussion to a tooth. Since the periodontal ligament is rich in proprioceptors and nociceptors, it reacts more actively to percussion than when the pain is coming from the dental pulp. The pulp is tested by the tooth's reaction to heat and cold, and by measuring the threshold of response in microamperes with a pulp tester.⁵³ Open contacts, periodontal disease, occlusal wear, missing teeth, fractured teeth, and fillings should be studied in relation to the occlusion.⁵⁷ Whenever acute trauma is a factor in the condition, the mandible and articular fossa should be thoroughly evaluated for fracture. Excessive wear of the occlusal surfaces for the age of the patient indicates the possibility of chronic bracing or bruxism, which is important in orofacial muscle pain. The teeth are evaluated for mobility by rocking a tooth back and forth. Mobility may occur in any direction. "Milking" with gentle massage of the gingiva toward the tooth may reveal the presence of pus in a periodontal pocket.

Pain may be referred to the head and temporomandibular joint by impacted molars. The onset of this pain is gradual and will spontaneously diminish in time. Radiographs, along with clinical examination, are necessary to form a diagnosis.

When there is positive therapy localization of a tooth and its gingiva that is not corrected with tooth manipulation as described in Chapter 9, the tooth

should be more thoroughly evaluated for disease. When pain is localized to the temporomandibular joint, the teeth can usually be ruled out as a cause unless they are painful. "In general this type of pain is not recurrent with long periods of freedom from pain, and it is rare without associated toothaches."⁴⁹

Ear

Visual examination of the external auditory canal and drum should be done for evidence of infection, tumors, and foreign bodies. "A normal ear drum and ear canal in the presence of normal hearing will rule out aural pathology with few exceptions."⁶⁷

Disturbance in ear function can result from temporomandibular joint disturbance. Its relation with remote problems is complicated since the ear is innervated by cranial nerves V, VII, IX, X, and the cervical plexus.⁴⁹ If the disturbance is from temporomandibular joint dysfunction, the condition is nearly always unilateral; the patient complains of pain, fullness, tinnitus, dizziness, and hearing loss. Arlen² blames much ear dysfunction on hypertonicity of the tensor palati and tensor tympani muscles. Both of these receive nerve supply from cranial V, helping to explain why there may be dysfunction of these muscles along with the muscles of mastication, also supplied by cranial V.

Nasal and Sinus Pain

Headaches associated with sphenoidal and ethmoidal sinus disease radiate behind the eyes and over the vertex of the skull. Pain may radiate to the back of the head, neck, and shoulders. Pain from frontal sinus disease is over the general frontal area. The maxillary sinus is grouped with antral disease, which radiates pain over the maxillary region, under the eye, along the zygoma, and toward the ear; it may also radiate into the forehead and posterior maxillary teeth. These headaches are less painful with decreased frequency. When in a supine position they are of a deep, dull, aching, non-pulsatile quality and are increased by shaking the head or assuming a head-down position. Straining, coughing, or wearing a tight collar, and other procedures that increase venous pressure, intensify the headaches.⁸ Frequently headaches related to sinusitis are, in reality, due to nasal allergy. The nasal mucosa has been found to exhibit a much higher pain sensitivity than the sinus mucosa.⁸

Sinus infection may result from roots of the upper teeth projecting into the maxillary sinus, where infection of the tooth spreads to the sinus. Pain radiation from tooth disease can simulate sinusitis;

the reverse is also true. (A complete discussion of the AK approach to sinusitis and nasal allergies is presented in Volume V of this series.)

Salivary Glands

The salivary glands can be involved with obstruction, infection, or neoplasm. There is local pain which may radiate to the facial area, and swelling follows. Pain radiated to the temporomandibular joint and ear is most commonly from the parotid glands. The swelling usually causes facial asymmetry. Normally the glands should palpate as very firm, non-tender, and non-painful. The orifices of Wharton's and Stensen's ducts should not have any redness or inflammatory change. A normal flow of saliva should be present on milking the glands and ducts. The key factor in differential diagnosis of salivary gland involvement from temporomandibular joint dysfunction is swelling, which is always present in salivary gland diseases.⁵

Herpes Zoster

Herpes zoster is characterized by the vesicular eruption and neuralgic pain along the skin of the peripheral sensory nerves affected. When the herpes results from involvement of the geniculate ganglion, there is usually pain in the ear and vesicular eruptions in the external auditory canal, on the auricle, soft palate, and anterior pillar of the fauces. When from the gasserian ganglion, the vesicular eruptions follow the ophthalmic division of cranial nerve V. Neuralgia may persist for years after the acute attack has subsided.⁵⁴

Myalgia

Dysfunction of the muscles of mastication may be reflected by intrinsic pain or radiation of pain to other structures. The radiation appears to be a result of trigger points as related by Travell⁶¹ and discussed later. Myositis or fibrositis is characterized by a dull, aching sensation in a muscle which increases as pressure is applied or the muscle becomes active. Much muscle dysfunction develops as a result of sustained contraction from bruxism, bracing, or an engram which is attempting to reduce the stress of malocclusion.

As previously mentioned, there has been much discussion in the literature of psychological factors as the etiology of temporomandibular joint dysfunction.^{31, 44, 50, 58, 69} This possibility should be thoroughly considered when there is evidence of bracing and bruxism and pain in the muscles of mastication, along with abnormal tooth wear. When bracing or bruxism is so severe and chronic that it leads to intrusion of the teeth, it may be the leading diagnostic

factor indicating a psychologic etiology. (See page 374 for more on bruxism.)

Neuralgia

The most common type of neuralgia in this area is tic douloureux (trigeminal neuralgia), which is relatively easy to differentiate from temporomandibular joint disturbance because of its characteristic, constant, classic findings.⁵¹ Its pain in the distribution of one or more divisions of the trigeminal nerve is severe, but intermittent. It is characteristically triggered by stimulation to the skin of the face or membranes of the mouth. The most common type is idiopathic, with no abnormal neurologic findings. Examination for and applied kinesiology treatment of trigeminal neuralgia are discussed in Chapter 5.

Glossopharyngeal neuralgia can affect the mandibular nerve in very much the same way as trigeminal neuralgia; it is difficult to differentiate the two. Sphenopalatine ganglion neuralgia is sometimes called a lower-half headache, since it involves the lower half of the face and never extends above ear level. The site of maximum pain is in the orbit and the base of the nose, and another location posterior to the mastoid process. The pain may radiate to other areas, such as the ear, neck, scapula, and shoulder.⁸

Vidian neuralgia is a closely related condition which refers pain to the nose, face, eye, ear, head, neck, and shoulder. The problem is typically unilateral and often nocturnal; it may or may not be associated with symptoms of nasal sinusitis.⁸ Headaches which appear to be related with these conditions often respond very well to applied kinesiology treatment of the total stomatognathic system. It is probable that the actual correction comes from improvement of cranial faults which are influencing the nerves, creating the neuralgia. Relief may also be due to improving the function of the sinuses and nasal mucosa.

Vascular Disease (Dysfunction)

Temporal arteritis is a localization of polyarteritis nodosa. The condition is a localization of an inflammatory process in the superficial temporal arteries. The characteristic of the headache is high intensity of a deep, aching, throbbing nature. Unlike most other vascular headaches, there is often a burning component. Some relief is obtained by being in an upright position, and the pain is somewhat reduced by digital pressure on the common carotid artery on the affected side. As in sinus conditions, bending over makes it worse. There may be pain on mastication; in some this may be the initial symptom. Usual laboratory findings are leukocytosis, averaging 12,000 to 13,000, elevated erythrocyte sedimentation

AK Examination and Treatment of the TMJ and Muscles of Mastication

rate, and a mild to severe anemia. There is common elevation of serum alkaline phosphatase, SGOT, and SGPT.⁸ The area over the artery evidences pain, heat, swelling, tenderness, and redness.

The migraine headache is actually a syndrome of widespread body disturbances. It is usually unilateral, lasting from a few minutes to several days. Contrary to popular opinion, a migraine headache can be minimal in intensity, but usually it is severe and associated with additional symptoms which may include irritability, nausea, photophobia, vomiting, constipation, and/or diarrhea. Pain is usually located in the unilateral temporal area, but it may radiate to the face and neck, and throughout the head bilaterally. The patient feels well, with an excess of energy prior to an attack and again after it. Patients can often recognize many prodromal signs of an impending headache, which may include visual disturbances, facial flushing or pallor, and vertigo.

The pain is often reduced significantly by pressure on the common carotid artery and the involved superficial artery.

The cluster headache is closely related to the migraine. It, too, is usually a unilateral pain, lasting up to two hours. The attacks tend to recur on a more frequent basis than migraine headaches. There may be one to several attacks per day, and these headaches may last for weeks or, less often, months. The series of headaches ends for no apparent reason, to return weeks or months later in another cluster. Alcohol is likely to induce headaches when a person is in a cluster period, but not during the stage of remission.

Vascular headaches often respond quite well to treatment determined by applied kinesiology examination. Greater description and the applied kinesiology approach are presented in Volume V of this series.

Therapeutic Methods

The therapeutic methods used to balance function in temporomandibular joint activity are the same muscular techniques used throughout the body in applied kinesiology. These include correction of the cranial-sacral primary respiratory mechanism, treatment to the muscle proprioceptors, spray and stretch and fascial release techniques, and sometimes muscle exercise. The cranial primary respiratory

mechanism is discussed thoroughly in Section I of this text; the other subjects are covered in Volume I of this series. It is necessary here only to discuss the subjects specifically as they relate to the stomatognathic system, prior to the examination of the temporomandibular joint, so that therapeutic methods may be referred to freely.

CRANIAL-SACRAL PRIMARY RESPIRATORY SYSTEM

As mentioned previously, it is important to determine the area of the stomatognathic system which is the primary involvement causing dysfunction. This is probably more true regarding cranial faults than any other area. If the cranium is primary and peripheral nerve entrapment is causing hypo- or hypertonicity of the masticatory muscles and hyoid — as well as other — muscles, it may be difficult or even impossible to return the secondary muscle dysfunction to normal with proprioceptive, spray and stretch, fascial release, or exercise therapies. If correction is obtained with these localized treatments and the cranial faults go uncorrected, the muscular disturbance will return, usually within a very short time. On the other hand, if muscular imbalance is contributing to the cranial fault, it will be difficult or impossible to obtain effective cranial

correction until there is localized treatment to the muscle. The muscles contributing to cranial faults can be those crossing a suture; in this case the muscle may either be hypo- or hypertonic, causing failure of support at the suture, or jamming. Most often the problem is a hypertonic muscle. Most of the muscles of mastication relate to cranial faults through their activity of pulling on cranial structures during mastication, forceful intercuspation, swallowing, etc. An exception is the temporalis, which must be considered with the leverage factor of the masticatory process as well as with those muscles crossing major sutures.

In this chapter we consider more closely the integration between temporomandibular joint function and the cranial primary respiratory mechanism. The neck flexors, extensors, and hyoid muscles must

be kept in perspective regarding their role in the total stomatognathic system. If cranial faults cause these muscles to receive improper nerve supply, the resulting imbalanced function may be the reason that cranial corrections are poor or quickly lost.

Another factor that may cause the cranium to be primary to temporomandibular joint dysfunction is the structural base that it provides for mandibular function. It is believed that cranial faults which cause an internal rotation of the temporal bone on one side, and an external rotation on the other, shift the position of the mandibular fossae to provide an imbalanced base for the condyles of the mandible.^{32, 33} Under these circumstances, the muscular imbalance that is present may be the body's attempt to bring the mandible into alignment with the imbalanced base of the cranium. What appears to be obvious dysfunction of the proprioceptors, trigger points, and fascial tension may be secondary to the muscular strain present because of the body's

appropriate response to a primary condition.

It is often necessary to examine and treat back and forth between the divisions of the stomatognathic system. Because of the great interaction in this system, it is often not possible to definitively determine what is primary and secondary. Hypothetically, if one could determine exactly what was present in the initial development of a condition, the correction of that primary factor would correct all subsequent disturbances. Unfortunately, the subsequent disturbance often tends to lock in the primary condition so that it cannot be effectively corrected. The physician knowledgeable in all areas of the stomatognathic system, as well as its interaction with the rest of the body, has an obvious advantage in obtaining correction of the total mechanism. It may be necessary to consult and recruit therapy from another profession, because there is no single group capable of correcting all of the many ramifications in this system.

PROPRIOCEPTORS

Direct treatment to the muscles of mastication is most often done in applied kinesiology by manipulation of the muscle proprioceptors. The most common involvement has been described by Goodheart^{17, 18} as dysfunction of the neuromuscular spindle cell, causing the muscles to be hypertonic and/or secondarily causing other muscles to dysfunction from inappropriate afferent stimulation. Although this has been the working hypothesis for successful clinical application, there is some question about whether this is the actual mechanism taking place. The hypothesis of neuromuscular spindle cell dysfunction was developed from what is thought to be manipulation of the neuromuscular spindle cell in larger postural muscles.¹⁶ In these larger muscles it is possible to manipulate the belly of the muscle over the location where there is suspected dysfunction of a neuromuscular spindle cell. This location is found by palpating for a nodular, swollen area which also shows positive therapy localization. By directing two points of digital pressure over the area, the muscle can be made to test strong or weak, depending upon the direction of digital pressure. If the two contacts are at the periphery of the palpable mass and are brought together, the muscle will test weak on manual muscle testing. If the two digital contacts begin close together over the center of the palpable mass and are drawn away from each other in alignment with the muscle fibers, a weak muscle will test strong. It is suspected that the manipulation affects the neuromuscular spindle cell by one or

more of the following methods: (1) manipulation breaks adhesions of the delicate septa between the intra- and extrafusal fibers, (2) it reduces edematous fluid within the nuclear bag area, or (3) it returns stretching capability to the nuclear bag of the intrafusal fibers. Since the neuromuscular spindle cell facilitates the homonomous, synergists, and fixators and inhibits the antagonists, there can be a wide range of dysfunction as a result of inappropriate afferent impulses from this receptor. Treatment of the neuromuscular spindle cell in one muscle will cause an inappropriately functioning remote muscle to function normally, as observed on manual muscle testing. This is called a reactive muscle, considered to be the one which receives no treatment but changes function as a result of treatment to a remote muscle. The reactive muscle is frequently a synergist, fixator, or antagonist to the muscle containing the dysfunctioning neuromuscular spindle cell, but it could be a remote muscle influenced by the primary one because of its neurologic connections in higher centers of the central nervous system. Support for the hypothesis is provided by Triano and Davis' electromyographic study of the influence on remote muscles from apparent neuromuscular spindle cell manipulation.⁶³

There is a question as to whether treatment is actually to the neuromuscular spindle cell in the muscles of mastication. First, it has not been consistently demonstrated that when the apparent neuromuscular spindle cell is manipulated the mastication

AK Examination and Treatment of the TMJ and Muscles of Mastication

tory muscles can be made weak or strong, depending on the direction of manipulation. This, of course, is because of the inability to directly test these muscles as is usual in manual muscle testing. In addition, on some of the smaller muscles such as the masseter and pterygoids, manipulation can only be done in one direction — linear with the muscle fibers. Goodheart has reported observing muscle strength change on a force transducer.²¹ More studies of this nature, along with electrophysiologic quantification, are needed to better understand the mechanisms taking place in these clinically effective procedures.

There has been some question of whether there are neuromuscular spindle cells located in the external pterygoid muscle. Godaux and Desmedt¹⁵ refer to two studies^{59, 65} which indicate that the jaw-opening muscles in the cat and in man do not contain any muscle spindles. However, neuromuscular spindle cells were found in the human external pterygoid muscles by Gill,¹⁴ located primarily in the middle third of the muscle. Other investigators have also found neuromuscular spindle cells located in the pterygoid and anterior belly of the digastric muscle.⁴¹ Clinical evidence indicates these muscles change function from muscle manipulation intended to influence the neuromuscular spindle cell. It may be possible that the treatment is actually a fascial release; perhaps it works through trigger points. In any event, the working hypothesis at this time is that the majority of treatment in applied kinesiology directed to the muscles is considered to be manipulation of neuromuscular spindle cells, which directly affect the homonomous and remote muscles.

There is considerable evidence that control of the masticatory muscles is mediated by afferent supply from the proprioceptors of the muscles and periodontal receptors. Dysfunction of this mechanism has been shown to relate with the TMJ syndrome. Bessette et al.⁴ demonstrated a significantly prolonged silent period in the masseter muscle following the jaw-jerk reflex in patients with TMJ syndrome. They classified the TMJ syndrome as (1) pain in one or both temporomandibular joints, (2) deviation or limitation in opening the mouth, and (3) the presence of TMJ sounds such as clicking or cracking in the intermediate ranges of mandibular

movement. Following occlusal equilibration, the silent period was reduced to that of normal subjects. Perry⁴³ found, in patients with occlusal disharmony and temporomandibular joint dysfunction, that low-grade activity of the temporalis and masseter muscles was exhibited on electromyography when the mandible was supposedly in the physiologic rest position. With correction of the condition, the muscles were inactive in the rest position. Griffin and Munro^{24, 25} have shown with electromyography that when there is temporomandibular joint dysfunction or potential dysfunction,³⁷ there is a change in the silent period after tooth contact in the open-close-clench cycle. There is also improper temporal muscle function, observed by electrical activity of the jaw-closing muscles during the opening phase of the cycle. This gives evidence that the function of reciprocal inhibition is not operating properly in these individuals as compared with those functioning normally.

The applied kinesiology examination procedures described later in this chapter appear to locate the proprioceptors in the complex which are sending inappropriate afferent information into the system, contributing to or causing malfunction. The inappropriate receptor stimulation appears to be in several categories; any of the following possibilities may exist. The neuromuscular spindle cell may be damaged on an acute basis, such as from prolonged stretching of a muscle during dental procedures, or it may be chronically under stress, such as in bracing, bruxism, or muscular imbalance from another disturbance in the stomatognathic system. The periodontal ligament receptors — either proprioceptor or nociceptor — may be sending improper information from unresolved body trauma, as occurs with a neurologic tooth condition. Another possibility is that the periodontal ligament receptors may be functioning normally, but they send information which creates confusion within the system because of malocclusion. The key factor in any of these possible etiologies of temporomandibular joint dysfunction is to locate the source of the afferent signaling which is in error, thus causing the problem. Applied kinesiology techniques appear to do this.

SPRAY AND STRETCH

The spray and stretch technique in applied kinesiology⁶⁶ is derived from Travell and Rinzler's trigger point technique.^{56, 60, 61} The usual indication to use this technique in applied kinesiology is the

weakening of a previously strong muscle on manual muscle testing immediately after it has been moderately stretched. This is called the muscle stretch reaction.¹⁹ Normally one would expect the muscle to

test stronger immediately after a moderate stretch because of the myotatic reflex.²² In most muscles of the stomatognathic system, direct manual muscle testing after moderately stretching the muscle is not possible, except on the sternocleidomastoid and upper trapezius. In other muscles, such as the masticatory, facial, and hyoid, it is necessary to use an indirect approach of stretching the muscle and testing an indicator muscle for weakening. Therapy localization to the muscle or complex in question may be added to the testing procedure. It is necessary that the therapy localization be negative prior to the muscle stretch, which is indicated by a previously strong indicator muscle not weakening when the point is touched. The muscle or complex of muscles is then moderately stretched and held in that position while the previously strong indicator muscle is tested for weakening. The muscle or muscle complex being tested for muscle stretch reaction can be stretched either actively or passively, but passive appears best. The speed of stretching and the amount of stretch can influence the test results.²⁰ A vigorous stretch may cause a false positive test since the normal reaction to vigorous stretching is muscle inhibition. The speed of stretching relates to the muscles' dominance of slow or fast fibers. Muscles dominant in slow fibers react to a slow stretch when there is a positive muscle stretch reaction, whereas muscles dominant in fast fibers react better to a fast stretch. The muscles of the stomatognathic system are dominant in fast fibers so the stretch should be rapid; care should be taken that it is not excessive when the full length of the muscle is reached.

To test the muscles of mastication for muscle stretch reaction, it is necessary to test the temporomandibular joint and muscle function for other factors first. This is explained later in this chapter when the total protocol of temporomandibular joint testing is presented; here it will suffice to discuss the muscle stretch reaction test. After the other tests have been completed and found negative or the condition corrected, the muscles are tested for muscle stretch reaction. Therapy localization is usually to the bilateral temporomandibular joints and should be negative at this point. The jaw is opened quickly but not forcefully to its maximum, and is held in that position while a previously strong indicator muscle is tested. Weakening of the indicator muscle indicates a positive test.

The muscles of mastication can now be evaluated for trigger points. There may be a positive muscle stretch reaction when no trigger points are present, since the muscle stretch reaction also indicates the need for fascial release technique, described next. When there is a need for either type of treatment —

spray and stretch or fascial release — the muscle will be shortened, as revealed by a positive muscle stretch reaction. The major differentiating factor is referred pain from trigger points.

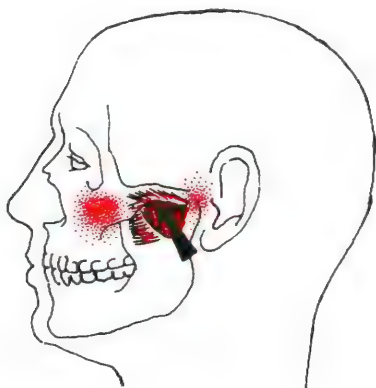
Trigger points can be located by palpating the muscle and its tendon. The location of the trigger point will be tender and, more important, it will radiate pain to a distant location. The referred pain pattern of trigger points is predictable. It has been mapped by Travell and Rinzler⁶⁰ and is shown in the accompanying illustrations for the muscles of mastication. Additional illustrations of trigger point patterns of the muscles of the stomatognathic system are in Chapter 16.

There are several methods for treating trigger points. The one generally used in applied kinesiology is spray and stretch.^{62, 66} This method is limited in the muscles of mastication to the masseter and temporalis. Other methods are digital pressure, dry needling, and injection of physiologic saline or a local anesthetic.⁶²

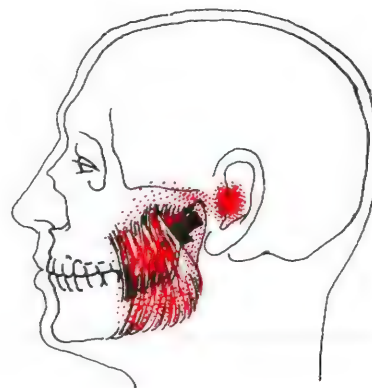
The technique of spray and stretch currently uses Fluori-Methane* spray as the vapocoolant. The muscle containing the trigger point is placed in mild passive stretch while the skin is sprayed from the trigger point to the area of referred pain. The mandible may be held open by the examiner, or a prop may be placed between the incisor teeth. The cylinder which protects the nozzle of the Fluori-Methane bottle during shipment is often the right size to use as a mouth prop, or it can be adapted by diagonally cutting it. The core from a two-inch roll of tape is the appropriate size for many individuals. The spray is applied in slow, even, uninterrupted strokes in only one direction, from the trigger point to the referred pain area. The sweeps of spray are applied approximately $\frac{1}{4}$ " to $\frac{1}{2}$ " apart until the total area is covered. The area should be covered only once and only in one direction, taking care not to overchill. Excessive cooling is not effective in breaking the neurologic pattern. When spraying the masseter and temporalis muscles, the patient's eyes should be protected. Prior to the development of Fluori-Methane spray, ethyl chloride was used. Fluori-Methane is preferable because it is non-flammable, non-toxic when used as described, and does not have as much cooling effect as ethyl chloride, which may be too great. Direct concentrated inhalation of Fluori-Methane must be avoided.

Digital pressure can be applied directly to the trigger point, which creates or intensifies the radiated pain. The digital pressure is held until the radiated pain subsides. This type of treatment has been called

*Gebauer Chemical Co., Cleveland, OH 44104



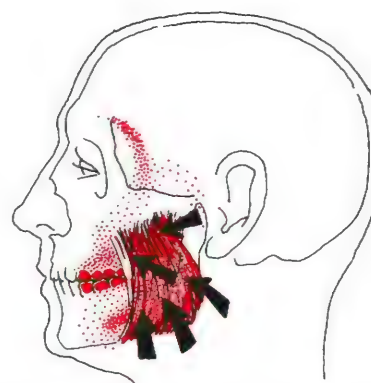
14—5. Composite pain reference pattern of the external pterygoid muscle.



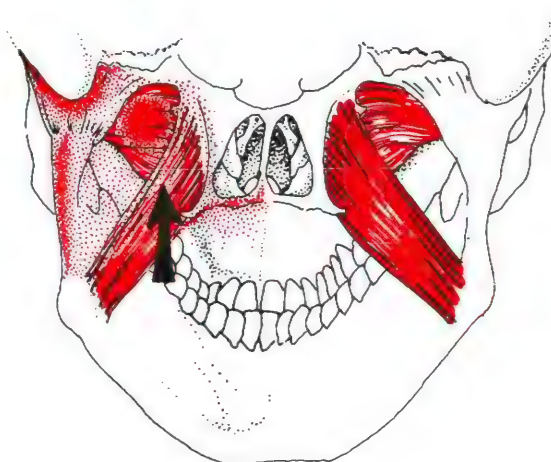
14—6. Deep layer of the masseter muscle pain reference patterns.

cellulome therapy,⁴² and is similar to the Japanese shiatsu treatment.³⁹ Accurate application is important. The vector of pressure on the trigger point should be changed until maximum radiation is observed. It is important to hold the pressure until a reduction in pain, either at the trigger point or in the referred pain area, is observed. The harder and more accurately the pressure is applied at the trigger point, the less time it will take for the pain to diminish. It may be necessary to repeat the treatment several times until the pattern is completely abolished. The length of time necessary for the pain to lessen during treatment gives some indication of the number of treatments that may be necessary.

Travell finds dry needling and physiologic saline injection of trigger points effective, but these are much more painful to the patient than injection of a local anesthetic into the trigger point.⁶² The injection approach may be needed in resistant trigger points located in the internal or external pterygoid muscle because these muscles cannot be treated with the spray and stretch technique; sometimes digital contact techniques are limited because of inaccessibility. In AK experience it is rarely, if ever, necessary to use needling techniques on the masseter or temporalis; the spray and stretch or digital contact method relieves the condition very well, as long as no other imbalances in the stomatognathic system are contributing to the trigger point. When trigger points seem to be effectively treated but continue to return, the total system should receive further evaluation.



14—7. Superficial layer of the masseter muscle pain reference patterns.



14—8. Internal pterygoid trigger point refers pain to the stippled areas.

AK Examination and Treatment of the TMJ and Muscles of Mastication



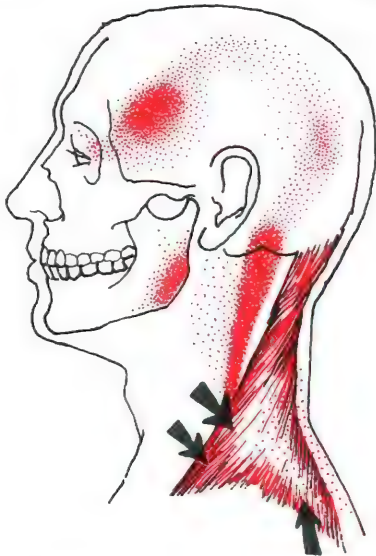
14—9. Anterior temporalis pain reference.



14—10. Middle temporalis pain reference.



14—11. Posterior temporalis pain reference.



14—12. Trigger points in upper trapezius and supra-scapular radiate pain to the stippled and solid areas.



14—13. Pain radiation from trigger points in the sternocleidomastoid muscle.



14—14. Spray and stretch technique for the masseter muscle. The muscle is stretched by the mouth prop. The spray should strike the skin at an angle.

AK Examination and Treatment of the TMJ and Muscles of Mastication

FASCIAL RELEASE

The need for fascial release technique is also indicated by the muscle stretch response described previously. A muscle requiring the treatment will be shortened and probably painful on digital pressure. There is no pain radiation from pressure at any location in the muscle, as there is from a trigger point. In a muscle which requires fascial release technique, there appears to be an inability of the fascia and muscle fibers to function together in an optimum manner. The muscular condition is described by Rolf,⁴⁸ and the treatment used in AK is very similar to hers as well as that of Nimmo.⁴⁰

Initial indication of the need for fascial release technique is a positive response to the muscle stretch examination, described with the trigger point technique above. As noted, the muscle will not have a trigger point causing referred pain to another area. This muscle will probably demonstrate a greater amount of shortening than the muscle with the trigger point. Treatment is simply deep massage parallel to the muscle fibers. This heavy pressure appears to break down adhesions between the fascia and muscle fibers and also appears to change the fascial fluids from gell to sol, as described in rolfing.⁴⁸ Nimmo⁴⁰ considers his receptor-tonus technique to be neurologic; the pressure is applied sequentially, released between applications, and applied again after a minute or two of rest. This appears to allow a neurologic adaptation to the pressure before another

pressure application. Possibly both hypotheses are applicable; no histologic or electrophysiologic studies have been found on either one.

The pressure applied should be relatively heavy, with care being given to the patient's tolerance. The muscle is often very tender, but subsequent pressure applications usually reveal a lessening of the pain. The pressure is applied over the full length of the muscle if possible, considering the anatomy of the particular muscle. Immediately after the fascial release technique is applied to the jaw-closer muscles, there is usually an increased range of motion; this is measured as the maximum interincisal distance. If there is pain during jaw motion, it will often be reduced. The applied kinesiology examination of muscle stretch reaction should be negative. Often only one treatment is necessary for permanent correction. If repeated treatment is needed, there should be further evaluation for bruxism, bracing, malocclusion, cranial faults, and other factors which cause muscle hypertonicity. Sometimes vitamin B₁₂, in combination with stomach and liver concentrate or nucleoprotein extract, helps prevent the condition from returning. When necessary, it is recommended three times a day for at least two or three weeks. A low dosage of B₁₂ — 5 micrograms — appears to work better than higher doses of B₁₂ alone. Vitamin B₁₂ without the stomach and liver factors does not appear to have any value on a clinical basis.

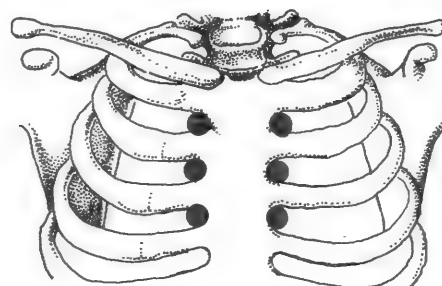
REFLEXES AND MERIDIANS

The reflexes and meridians are not treated as frequently for the muscles of mastication as for some other muscles throughout the body. All the muscles of mastication share the same reflex locations, stress receptor, and meridian. When involved, they are treated the same as for other locations within the body. Methods of treatment are described in Volume I for the reflexes and stress receptors, and in Volume III for the meridian system.

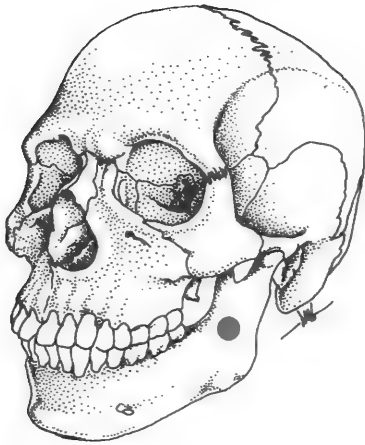
Neurolymphatic:

Anterior: 1st, 2nd, and 3rd intercostal spaces adjacent to the sternum.

Posterior: Laminae of T2, 3, and 4.

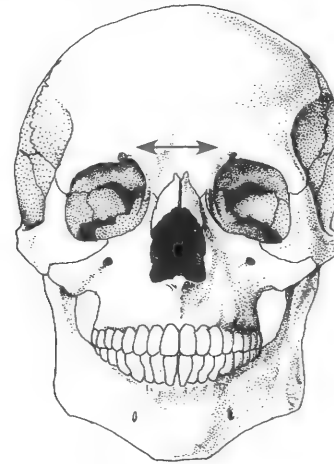


14—15. Neurolymphatic reflexes for muscles of mastication.



14—16. Neurovascular reflex.

Neurovascular: Located in the center of the ramus of the mandible.



14—17. Stress receptor.

Stress Receptor: Located approximately 1" above the glabella in the transverse plane.

Meridian: Stomach.

Applied Kinesiology TMJ Evaluation

The examination described in this section is typically referred to as TMJ evaluation. It examines the interaction of the muscles of mastication, but it has a much broader scope in evaluating many aspects of the stomatognathic system; this is necessary because of the great amount of interaction taking place within this system. For example, the muscles of mastication may have improper temporal action, but the improper timing sequence may not be due to muscle dysfunction; it may be secondary to malocclusion or a neurologic tooth involvement. The examination and treatment sequence is directed primarily to the temporomandibular joint and the muscles of mastication, but it leads to many other areas of dysfunction within the stomatognathic system.

The tests used to evaluate temporomandibular joint motion have been developed primarily by Goodheart^{17, 18} and are mostly indirect. Various parameter changes are made testing the temporomandibular joint, its muscles, the teeth, and other aspects of the stomatognathic system; the effect is observed on a previously strong indicator muscle such as the tensor fascia lata, psoas, neck flexors, deltoid, etc. Generally, observation is for weakening of an indicator muscle. Not all indicator muscles will

weaken in every malfunction in the stomatognathic area. In some cases only "problem muscles" or those indicated on the TS line will show a positive finding. Occasionally a remote muscle associated with the problem may strengthen as a result of test procedures directed to the stomatognathic system.

These clinical approaches to examination have been supported by predictable, confirming muscle findings indicated by a testing procedure. For example, when a positive test for muscle stretch reaction is found, it will consistently be confirmed by finding one or more of the muscles indicated by the test to be hypertonic and in need of trigger point or fascial release technique. When appropriate treatment is applied, the positive indications of the examination will be abolished.

The following procedure is an organized approach to evaluate the muscles and function of temporomandibular movement. It is one way of making an efficient and comprehensive evaluation of the stomatognathic area. The temporomandibular joint and related functions are evaluated with and without therapy localization to the TMJ. First we will discuss no therapy localization to the TMJ. The testing procedures will be directed toward centric occlusion, motion of the jaw, and forced jaw movements.

NO THERAPY LOCALIZATION TO THE TEMPOROMANDIBULAR JOINT

CENTRIC OCCLUSION

The first testing procedure is to have the patient place his teeth into centric occlusion; then the examiner tests a previously strong indicator muscle for weakening. This is done first with relaxed tooth contact and then by biting hard. It is best to ask the patient to put his back teeth together without biting hard. When some patients are asked to put their teeth together, they only occlude the anterior teeth. This is usually done by individuals who contact first on the anterior teeth and slide posteriorly into centric occlusion. If no weakening of the indicator muscle is found, then the patient is asked to bite hard, which is forced centric occlusion.

If there is a weakness of an indicator muscle when a patient occludes his teeth in either of these tests, other factors of an examination may be led astray. A patient may inadvertently bite down while the physician is evaluating some other area of the body and cause the muscle being tested to appear weak. This is eliminated when the physician is aware of the stomatognathic area's influence on total body function.

Relaxed Centric Occlusion

When an indicator muscle weakens in relaxed centric occlusion, suspect a neurologic tooth involvement (page 265), malocclusion (Chapter 12), or reduced vertical dimension (page 370).

The first consideration should be for a neurologic tooth since it is the simplest to correct and does not require irreversible procedures. A neurologic tooth can be most easily located by therapy localizing broad areas of the dental arch until an area tests positive; then individual teeth are therapy localized until the precise one is found. The tooth should be challenged in all directions, including both axial directions. When a positive challenge is found, determine the phase of respiration which abolishes it; manipulate the tooth in the direction of the positive challenge on the phase of respiration which abolished the challenge. After manipulation the tooth should again be therapy localized. The occlusion should also be tested in a relaxed position to make certain there are no additional receptor involvements as a result of the neurologic tooth.

If an indicator muscle weakens during relaxed centric occlusion but there is no evidence of a neurologic tooth, malocclusion or diminished vertical dimension will probably be found. At this stage of examination it is too early to definitively attribute the

weakening to either of these conditions. Later in the examination cranial faults, imbalance of the muscles of mastication, hyoid muscle involvement, etc., may be found which, when corrected, also correct the malocclusion or change the evidence of diminished vertical dimension.

The applied kinesiology test for diminished vertical dimension can be done at this time, but no final conclusions should be made. The vertical dimension is increased and the indicator muscle which weakened during relaxed centric occlusion is re-tested to determine if it regains its strength on manual muscle testing. Vertical dimension is usually increased by placing tongue depressors between the molar teeth on both sides. If the indicator muscle does not now weaken, one of two variables has influenced it. It could be the increase of vertical dimension, or it could also be the elimination of noxious periodontal ligament proprioceptive stimulation as a result of malocclusion. Often no change in the indicator muscle occurs when single tongue blades are placed between the molar teeth on either side. In this case, increase the vertical dimension more by placing two tongue blades between the molar teeth on each side. This process is continued until the amount of vertical dimension increase needed to eliminate the positive weakening of the indicator muscle is found. Quite often when vertical dimension is increased beyond the level where an indicator muscle becomes strong, the muscle will weaken again. At this stage of examination and correction, elimination of the indicator muscle weakness by increasing vertical dimension means only that this vertical dimension is in harmony with the muscles, cranium, and other aspects of the stomatognathic system in their current status, which may not be correct. Correction of additional abnormal function later in the examination and treatment may change the total balance so that what appears to be a correct vertical dimension at this time is no longer appropriate.

Forced Centric Occlusion

When there is no weakening of an indicator muscle on relaxed centric occlusion, have the patient bite down hard in a forced centric occlusion. Test a previously strong indicator muscle for weakening while the patient continues to bite down hard. If the indicator muscle weakens, the possibilities are that there is a neurologic tooth involvement, malocclusion, cranial faults, muscles of mastication imbalance,

or other more remote structural problem such as cervical spine dysfunction.

The possibility of a neurologic tooth is basically the same as discussed under "Relaxed Centric Occlusion." The only difference is that a greater amount of force is required to exhibit the positive finding. The dentition is evaluated for a neurologic tooth as described above and on page 265. Malocclusion should not be considered at this stage for the reasons previously mentioned.

Ideally, all cranial faults have been corrected prior to this point in the examination. Their possible presence when an indicator muscle weakens with forced centric occlusion relates either to malocclusion or to the way the muscles of mastication are pulling on the cranium.

As described in Section I, normal occlusion enhances cranial motion and balance because the muscles of mastication pull on important levers of the cranial bones which activate their motion. Forced centric occlusion tends to balance the cranium if there is a good occlusion, because the mandible — being a single bone — tends to form the maxillary arch and adjust the maxillae to a balanced form. If the cranial mechanism is locked, the maxillae may not be able to shift from a distorted position to enable the teeth of the upper and lower dental arches to intercusate correctly. In this event, a previously strong indicator muscle will weaken on forced centric occlusion. There is an actual malocclusion because of the distorted upper arch, but it is secondary to an imbalance of the cranial primary respiratory mechanism. It is possible under these circumstances for a dentist who is not knowledgeable about the cranial primary respiratory mechanism to observe the malocclusion and equilibrate it so that the occlusion matches the cranial dysfunction and locks in that condition. The cranium needs to be thoroughly evaluated for possible cranial faults. It should be emphasized that a thorough investigative effort should be done to find cranial faults **before equilibration**. Under the circumstances described here, cranial faults are very often present. Various vectors of challenge should be used, along with respiratory assistance and therapy localization as described in Section I. Evidence of cranial disturbance can often be found by therapy localizing to a pterygoid process. When cranial faults are present, this area will frequently show positive therapy localization when other areas related with cranial faults do not. This is because of the importance of the sphenoid bone in cranial faults; its pterygoid plates will nearly always be in stress with the pyramidal process, especially if there is malocclusion.

If no cranial faults are found, weakening of an

indicator muscle on forced occlusion is probably due to intrinsic masticatory muscle dysfunction; that is, they are not dysfunctioning secondarily to improper nerve supply from cranial faults. When there is imbalance of the masticatory muscles, they may pull on the levers of the cranium to distort it and cause an indicator muscle to weaken. Usually the muscular condition is that of imbalance between the two sides. Later in the examination these muscular imbalances will be revealed. When they are corrected, forced occlusion should be re-tested to be certain it is negative.

JAW MOVEMENT WITHOUT TL

Testing a previously strong indicator muscle while the patient is moving the mandible to open or close his jaws quickly appears to test the fast fibers of the muscles of mastication. This hypothesis has developed as a result of pantothenic acid eliminating the positive finding. Pantothenic acid is the nutrition used in applied kinesiology for fast muscle fibers in aerobic and anerobic muscle function (see Volume I). If a strong indicator muscle weakens when a patient slowly moves the mandible, it indicates the slow fibers are involved. This is neutralized by chewing low potency chelated iron.

When testing for apparent fast muscle fiber dysfunction in the muscles of mastication, care must be taken that the patient moves his jaw rapidly but does not occlude the teeth or stretch the jaw wide open. Either situation adds other factors to the test, such as periodontal ligament receptor stimulation, muscles pulling on the cranium, ligament and muscle stretching, etc.

JAW FORCED OPEN WITHOUT TL

When the jaw is forced open without TMJ therapy localization and an indicator muscle weakens, it indicates that the jaw-closing muscles are hypertonic or there are cranial faults. The jaw may be forced open either actively or passively. Passive opening gives the most clear-cut findings because active opening requires contraction of the external pterygoid and anterior belly of the digastric muscle. A patient can passively open his jaw by contacting the chin at the symphysis menti area and stretching the jaw. First it should be determined that there is no positive therapy localization when the patient touches his chin. The ideal method is for a support person to hold the jaw open while the physician tests the indicator muscle. Later there is discussion of holding the jaw in various positions to test specific muscles.

AK Examination and Treatment of the TMJ and Muscles of Mastication

When all of the testing procedures are familiar, many can be combined. In the absence of a support person the physician can hold the jaw open; however, it is not as convenient and can sometimes be quite difficult. Care must be taken that the muscle testing is done accurately, since the physician holding the jaw open is very limited in his ability to correctly stabilize the patient. If there is a positive finding on forced opening when done actively but not passively, one should evaluate the external pterygoid as indicated later, or the anterior belly of the digastric as presented in Chapter 13. There may be a reactive muscle condition.

Hypertonic Closing Muscles

Hypertonic closing muscles, evidenced by an indicator muscle weakening, indicate one or more of the muscles has a trigger point and/or fascial release technique is needed. If the patient experiences a lot of pain, the trigger point is most likely; if there is considerable reduction in jaw opening, manipulation of the fascia is probable. Prior to treatment it is valuable to record the maximum interincisal opening for comparison after treatment. The amount of opening will usually be greatly increased following either the trigger point or fascial release technique.

Trigger points are most easily located by observing the referred pain pattern as indicated in the illustrations on pages 418-419. Palpate the muscle relatively lightly at first, looking for a painful area that refers remote pain. If the trigger point is found in the masseter or temporalis muscle, the spray and stretch technique is generally used. When in the internal pterygoid, the digital pressure technique is

used. As mentioned previously, it may be necessary to needle the trigger point, with or without an anesthetic.

Fascial release is accomplished simply by a broad, sweeping digital pressure applied parallel to the muscle fibers. Whether searching for a trigger point or applying fascial release, it is important to cover as much of the muscle as possible. In the muscles of mastication, ability to digitally contact the entire muscle is limited (see muscle palpation earlier in this chapter).

Cranial Faults

Weakening of an indicator muscle from forced jaw opening may occur from the muscles' pull on their lever attachments, influencing cranial faults. This, in essence, is the same as challenging the cranium. When the cranium functions normally, there is no adverse influence from various pressures or traction of muscle function. As discussed in Section I, maximum jaw opening activates the normal cranium, and repeated maximum stretching even activates the cranium which is functioning abnormally. The cranium should be thoroughly evaluated for cranial faults if there is weakening of an indicator muscle on forced opening and no hypertonic muscles are found. Sometimes the indicator muscle will weaken even after hypertonic muscles have been treated. This indicates a dual problem, including both muscle dysfunction and cranial faults. Probably the hypertonic muscles developed as a result of improper nerve supply secondary to cranial faults.

THERAPY LOCALIZATION TO THE TEMPOROMANDIBULAR JOINT

Further examination of the temporomandibular joint is done with the patient therapy localizing to the joint, either on both sides or individually. Care must be taken that the patient is actually therapy localizing to the temporomandibular joint. When requested to touch the jaw joint, a large percentage of patients will therapy localize considerably below the articulation. When therapy localizing bilaterally, either one or both sides may be involved. With therapy localization to one side only, positive findings indicate that the muscle(s) or some other structure is involved on that side. Various testing parameter changes will be made with the patient therapy localizing to the TMJ. First, simply therapy localize with no movement of the temporomandibular joint. Following will be various jaw movements in combination with therapy localization.



14—18. Care should be taken that the patient is touching directly over the TMJ during therapy localization.

TL TO THE TMJ — NO MOVEMENT

The first test done with therapy localization to the temporomandibular joint is to test an indicator muscle for weakening when the patient simply therapy localizes to the TMJ. Weakening of an indicator muscle under these circumstances indicates pathology in the TMJ or adjacent tissues, or disc dysfunction; differential diagnosis is necessary to delineate what is involved. The temporomandibular joint does not seem to demonstrate subluxation as ordinarily interpreted in chiropractic. The joint proprioceptors do not seem to be stimulated with joint misalignment the same as other articulations of the body. Subluxation of the temporomandibular joint relates more to disrelation of the disc with the condyle.

Disturbance of the TMJ disc will not always be evidenced by positive therapy localization. Popping and cracking on articular motion are almost always indicative of disc dysfunction. Crepitation may be associated with disc dysfunction, but it may also relate with degenerative or other joint diseases. As discussed in Chapter 11,³ disc displacement means that the disc is not relating properly with the condyle, whereas disc dislocation means that the disc is anterior to the condyle and is completely disrelated. This often causes an opening lock which may or may not respond to a conservative approach.

When disc manipulation and mandibular anterior repositioning are responsible for increased jaw opening, improvement is rapid because the disc is no longer locking the condylar translation (see Chapter 12). It bears repeating that gradually increased jaw opening is not necessarily a sign that the disc condition is improving. Gradually increased opening very often indicates that the posterior attachment of the disc is stretching and becoming more compromised. When there is severe disc dislocation with loss of posterior attachment, surgical procedures are available to reposition the condyle and re-establish the posterior attachment.^{12, 35}

Whenever therapy localization of the temporomandibular joint is positive without movement, consider the differential diagnostic factors presented earlier in this chapter. Often the positive therapy localization is due to disc dysfunction; however, there may be a combination of disc dysfunction and pathology. Physical diagnosis of the joint and the adjacent tissues, along with radiographs and laboratory diagnosis, can quite often define the reason for the positive therapy localization. If disc dysfunction is the diagnosis, conservative care often rather quickly abolishes the positive therapy localization, usually immediately after disc manipulation.

ISOMETRIC CONTRACTION WITH THERAPY LOCALIZATION

Testing the muscles of mastication under isometric conditions can be accomplished by the physician holding the mandible in a jaws-closed position while the patient attempts to open against the resistance.²¹ This is done while the patient therapy localizes to the TMJ, and a previously strong indicator muscle is tested for weakening. The test is also done with the mandible held in a jaws-open position, and the patient attempts to close his jaws while the physician resists the motion. Weakening of a strong indicator muscle under either condition appears to indicate that the muscle(s) involved needs strengthening. This is usually accomplished by manipulation of the neuromuscular spindle cell. Attention may need to be given to the neurolymphatic and neurovascular reflexes, stress receptors, or meridian balancing.

JAW MOVEMENT IN SAGITTAL PLANE WITH TL

Much of the involvement of the masticatory muscles appears to be a type of reactive muscle condition, evidenced by an indicator muscle weakening under different sequences of jaw movement. First a screening test is done in each major plane of movement; this is valuable for quickly evaluating the muscles of mastication. If positive, further testing delineates which muscle(s) must be treated.

For this testing, the supine patient's head and neck should be in basically a neutral position, without excessive flexion which is often caused by the headpiece of a chiropractic table, or excessive extension as is often seen on dental chairs. The "V" type head rest found on some dental chairs can place forces into the cranial mechanism which may change the results of the testing procedure.

Muscle activity in jaw movement in the sagittal plane is evaluated by having the patient therapy localize to the temporomandibular joints while simultaneously opening and closing his jaw, as in the previous testing procedure without TL. Again, take care that the patient does not click his teeth together or excessively stretch the muscles with forced jaw opening. Weakening of an indicator muscle in this situation indicates that the muscles are not functioning in harmony with each other. The treatment which abolishes this finding indicates that the disturbance is in the nature of a reactive muscle as seen in applied kinesiology.^{18, 66}

Sometimes it is questionable whether the patient is concentrating adequately on contracting the

AK Examination and Treatment of the TMJ and Muscles of Mastication

indicator muscle while at the same time opening and closing the mandible. This can quickly be clarified by simply having the patient discontinue therapy localization, and the examiner re-tests the indicator muscle while the patient continues to open and close his jaws. The muscle should now remain strong, whereas it previously weakened with therapy localization. The only exception is if the patient is involved with the fast or slow fibers and needs pantothenic acid or iron chelate as described earlier. The fast or slow fiber disturbance is not nearly as frequent a finding as weakening of an indicator muscle while therapy localizing the TMJ with jaw movement in the sagittal plane. When this screening test is positive, the next step is to determine whether the opening or closing muscles are primary, and on which side.

Determining whether the opening or closing muscles are involved is accomplished by having the patient stop the jaw motion in either the open or closed position. This can be done at the end of a series of opening and closing activity by simply having the patient open and stop in that position, or open and close and stop in that position. Again, care must be taken that the patient does not force open, stretching the muscles, or click the teeth together into intercuspation at the end of closing. Opening should be to the relaxed, fully open position without strain. Closing should approximate the rest position. When an indicator muscle weakens after the patient stops in the open or closed position, the last muscles used appear to be involved. Thus a positive finding on opening indicates the external pterygoid and possibly the anterior belly of the digastric need treatment; the platysma is rarely involved. When the indicator muscle weakens after the patient stops in the closed position, the masseter is probably the primary involvement, with the possibility of the temporalis and internal pterygoid needing treatment.

After determining whether the condition is of the opening or closing muscles, the side of involvement is then identified. This is accomplished by repeating the opening or closing maneuver, but therapy localizing only to one side. Usually an indicator muscle will weaken with therapy localization on one side only, but the involvement can be bilateral.

Mandibular Opening Muscles

If the positive finding is in

the open position, the external pterygoid probably needs treatment. Sometimes the anterior belly of the digastric muscle is involved. Rarely are both dysfunctioning coincidentally. When these muscles are not found positive with therapy localization, the platysma should be evaluated.

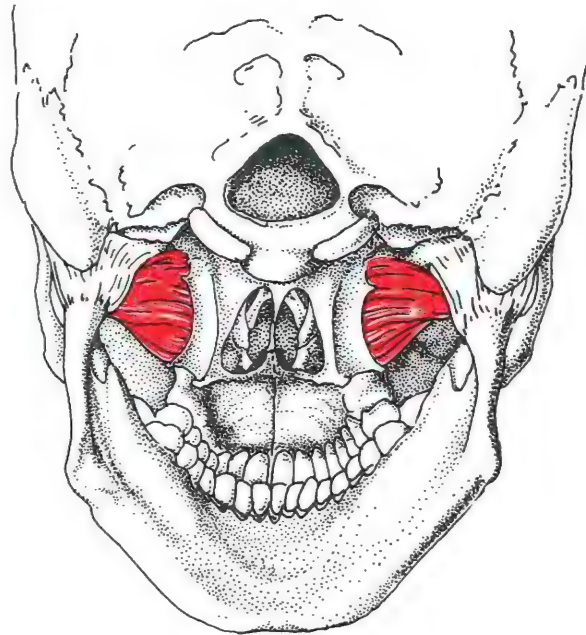
When the positive muscle is found, treatment appears to be to the neuromuscular spindle cell. There will be positive therapy localization over the area requiring therapy. Manipulation of the spindle cell is digital pressure drawn parallel with the muscle fibers over the area which therapy localized. The double directional manipulation generally used in spindle cell treatment is not possible because of the smallness of these muscles. Immediately after treatment, the mandibular movement with therapy localization should be negative. If it is not, re-evaluate and treat the muscle or another opening muscle — external pterygoid or digastric; rarely the platysma — which has positive therapy localization.

As noted above, when therapy localization is positive with the jaw stopped in the open position, the most common involvement is with the external pterygoid muscle. Usually it is treated within the oral cavity. The location can be found by having the patient therapy localize to the muscle inside the mouth. The physician must observe and direct the patient to the proper location, which requires accurate knowledge of anatomy. In this case it is difficult to palpate the swollen fibrous tissue associated with a dysfunctioning neuromuscular spindle cell.

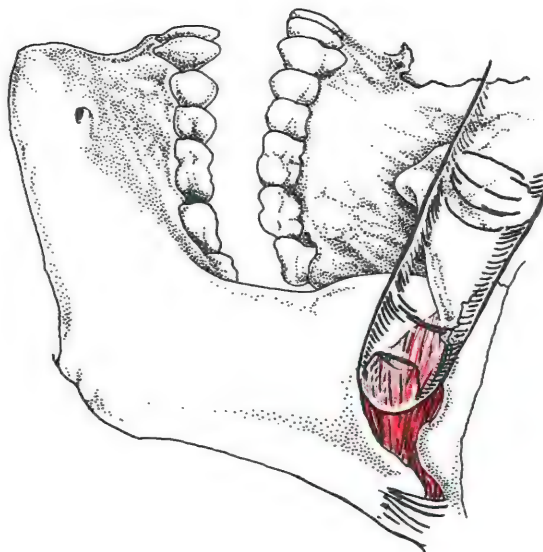


14—19. Digital contact to lower belly of the external pterygoid muscle (see also figure 14—3).

The physician then contacts the muscle posterior to the area of positive therapy localization and slides his finger anteriorly, parallel with the muscle fibers. This is usually very uncomfortable to the patient; it should be done quickly and efficiently. Sometimes the area of positive therapy localization is external. Nimmo⁴⁰ directs pressure toward the external pterygoid through the buccinator. With this approach it may be possible to influence the muscle closer to its insertion. Because the external pterygoid lies deep to this approach, contact is rather limited at this point; however, it is sometimes the key factor that obtains balance in the muscles of mastication. The location is immediately anterior to the condyle, between the zygomatic process of the temporal bone and the mandibular incisure. Slight opening of the jaws makes the muscle more accessible by lowering the mandibular incisure. Care must be taken not to open the jaws too wide as the condyle moves forward, obliterating the access space.



14—20. Posterior view of external pterygoid muscle.



14—21. Note the external pterygoid lies deep to the external surface. Sometimes external contact as described by Nimmo⁴⁰ is effective in improving the muscle's function.

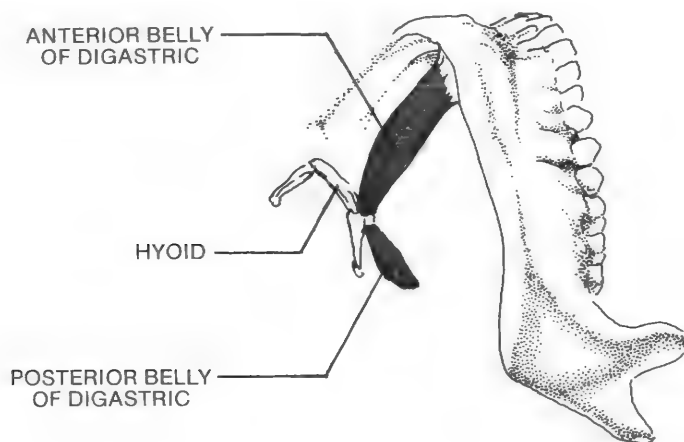


14—22. External contact to influence external pterygoid muscle.

AK Examination and Treatment of the TMJ and Muscles of Mastication

When there is a positive finding of involvement of a jaw-opening muscle but the external pterygoid does not show positive therapy localization, the anterior belly of the digastric should be evaluated. This muscle is easily located in comparison with the external pterygoid. It is evaluated with therapy localization and palpation for apparent neuromuscular spindle cell dysfunction. Treatment is digital

pressure over the area of positive finding, parallel with the muscle fibers. It does not seem to make any difference in which direction the pressure is applied. Often when this muscle is found positive with a jaw-opening problem, it will also be positive on hyoid challenge as described in Chapter 13. Treatment for both conditions is identical.



14—23.



14—24. Therapy localization to the anterior belly of the digastric. When therapy localizing to the small muscles, use a single finger to help localize the involvement.



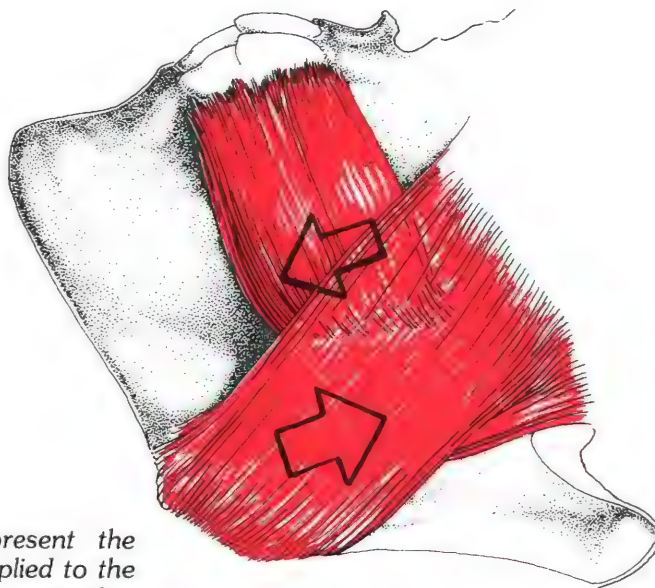
14—25. Treatment is apparently to the neuromuscular spindle cell to weaken the muscle.

Mandibular Closing Muscles

If the positive finding is in the jaw-closing position, the masseter, temporalis, or internal pterygoid muscle(s) needs treatment. Again, the approach is apparently to the neuromuscular spindle cell, which is located by therapy localization. In the masseter and temporalis it can be readily palpated as a nodular or swollen fibrous-like structure in the belly of the muscle. This palpatory finding is usually felt more easily in the masseter muscle than anywhere else in the body.

Goodheart's original description was combined treatment to the masseter and buccinator muscles simultaneously.¹⁸ The physician contacts each muscle with a thumb, the more important contact being the thumb which glides over the neuromuscular spindle

cell in the masseter. It often helps to lubricate the patient's skin with lotion prior to treatment so the thumbs will glide smoothly over it; this is particularly important in a male who has whisker stubble or a beard. The patient maintains jaw closure during treatment. The physician places one thumb at the inferior aspect of the area which therapy localized and was identified by palpation; the other thumb is placed at the upper border of the buccinator. Therapeutic motion is a scissors-like action of the two thumbs coming together and passing each other approximately one-half inch apart on the average-sized patient. The pressure applied is relatively hard, but not so great as to cause tissue trauma or adverse muscle reaction.



14—26. Arrows represent the direction of force applied to the masseter and buccinator muscles.

AK Examination and Treatment of the TMJ and Muscles of Mastication

14—27. Therapy localization for masseter dysfunction. There is usually an easily palpated nodular and fibrous area where the apparent neuromuscular spindle cell dysfunction is.



14—28. The initial contact for masseter-buccinator manipulation is at the inferior aspect of the masseter and at the top edge of the buccinator.



14—29. The treating thumbs move in opposite directions in a scissors-like motion to travel the length of the masseter and across the buccinator.

If no positive evidence of a dysfunctioning neuromuscular spindle cell is found in the masseter, or if there is still positive evidence of a closing muscle dysfunction after treating the masseter, evaluate the temporalis muscle. The area of dysfunction may be found in any division of the temporalis muscle. When the disturbance is related with the closing test, the positive therapy localization and palpatory indices will usually be found in the anterior or middle belly. If there is associated disc dysfunction, the problem will

usually be found in the posterior belly. The therapeutic approach is to weaken the muscle with neuromuscular spindle cell manipulation. In this larger, fan-like muscle, the routine of two-point digital pressure directed toward the center of the neuromuscular spindle cell can be used. This muscle, heavily covered with fascia, often requires a slightly heavier therapeutic pressure for effective results than do the other muscles of mastication.

JAW MOVEMENT IN TRANSVERSE PLANE WITH TL

Testing procedures with jaw movement in the sagittal plane should be the first tests done in evaluating the muscles of mastication; they will find the majority of involvements. The procedures from this point on will progressively find more unusual and covert muscular dysfunction. The first two examination modes in the following material activate the same muscles of mastication previously evaluated with sagittal jaw movement, but in a different sequence. To thoroughly understand these examinations, it is necessary that the physician be familiar with the anatomy and actions of the muscles of mastication. It might be advisable to review the muscle actions that have been determined by electromyography, which were presented in Chapter 9 with the anatomy of the muscles of mastication. Not all possible muscle involvement will be included in this discussion. The prime jaw movers of each action will be listed. In the occasional situation where there is a positive finding but no involvement of the muscles associated with the movement and discussed here, the physician should consider the secondary muscles active in the movement, such as the anterior temporalis.



14—30. Therapy localization with jaw in transverse movement.

Non-forced Transverse Movement

When testing jaw motion in the transverse plane, a procedure similar to that which was done with sagittal jaw movement is followed. The only difference is that the jaw is wagged from side to side while the patient therapy localizes to the TMJ and the examiner tests a previously strong indicator muscle. The comment, "Wag your jaw from side to side while

you touch your jaw joint," is a good way to describe this action to the patient. He soon associates open and close with the sagittal plane, and wag with the transverse plane. It is usually not necessary to be concerned about whether the patient touches his teeth or opens his jaw too wide; correct jaw activity is usually automatically assumed. It is necessary to be careful that the patient does not move his jaw laterally to such an extent that the muscles are forcefully stretched or contracted. Forcefully stretching the muscles can change the parameter of the test to that of a muscle stretch reaction, or the muscle may pull on the cranium and challenge for cranial faults.

The first procedure is for the patient to wag his jaw back and forth continuously while an indicator muscle is tested. This is a screening test which, if positive, indicates further testing in this mode. If an indicator muscle weakens, the next step is to begin delineating which muscle(s) is involved.

When the anterior portion of the jaw moves laterally, the prime movers are the ipsilateral posterior temporalis and the contralateral external and internal pterygoid. Step two is done to begin determining which muscle(s) is involved. Again the patient moves his jaw from side to side in the same manner, but he stops with the jaw in the lateral but not forced position; an indicator muscle is tested for weakening. Involvement of the masticatory muscles will be related to the side on which jaw movement stopped. If positive, the ipsilateral posterior temporalis or contralateral internal or external pterygoid muscle is probably dysfunctioning. Usually there will be positive weakening of an indicator muscle only when the jaw is stopped either to the right or to the left when both temporomandibular joints are therapy localized. The next step is to determine whether the muscle involvement is on the ipsilateral or contralateral side to jaw movement. This is done by therapy localizing only one TMJ and repeating the side-to-side maneuvers, stopping with the jaw on the side that was previously positive with bilateral therapy localization. If the test is positive when therapy localization is on the side of lateral jaw movement, the posterior temporalis is indicated. If positive with therapy localization on the opposite side of jaw movement, the internal or external pterygoid muscle is dysfunctioning. It is possible for both muscles to be involved, but that is not usually the case.

An example will show how quickly these tests can be accomplished. We begin the example with the physician having observed the weakening of an indicator muscle as the patient moves his jaw from side to side while maintaining bilateral TMJ therapy localization. The physician says, "Stop with your jaw

AK Examination and Treatment of the TMJ and Muscles of Mastication

to the right.” There is no weakening of the indicator muscle. “Wag your jaw again and stop to the left.” There is weakening of the indicator muscle, which indicates that the left posterior temporalis or the right internal or external pterygoid muscle is involved. “Now take your right hand off your jaw joint. Wag your jaw again and stop to the left.” There is no weakening of the indicator muscle. “Now put your right hand on your jaw joint and take the left off. Wag again and stop to the left.” The indicator muscle weakens, indicating involvement of the right internal or external pterygoid muscle.

After determining which muscle(s) is involved by the above active testing procedure, further delineation by therapy localization and palpation is done to confirm or further determine which muscle(s) is involved. Continuing the example above, the right internal and external pterygoid muscles are therapy localized. Positive therapy localization is found inferior and lateral to the pterygoid process which is the location of the internal pterygoid belly indicating that muscle is positive. The physician's palpation finds this location to be exquisitely tender. Treatment to the muscle is similar to that described previously for the muscles of mastication. A sweeping contact is made over the area of dysfunction parallel with the muscle fibers. Care must be taken that the pressure is not too great for the patient's tolerance or hard enough to cause trauma to the muscle. It may be necessary to wait a minute or two for sequential treatment. If this is necessary, it will probably be noted that there will be less tenderness on the second therapeutic effort. Re-test with therapy localization and jaw motion with TMJ therapy

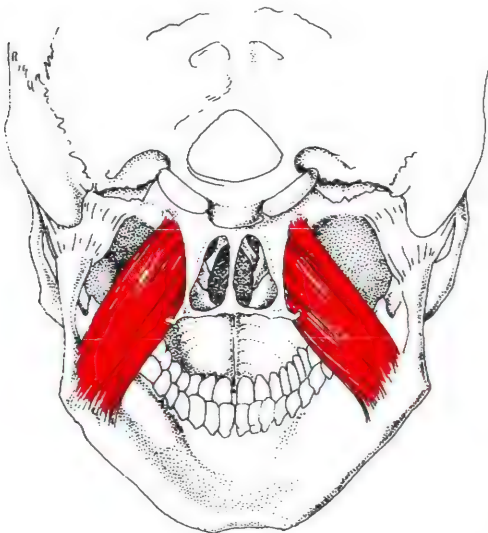
localization to confirm successful treatment. Indication to repeat the therapeutic maneuver is failure to eliminate positive therapy localization to the muscle or weakening of an indicator muscle with jaw movement and therapy localization.

Forced Transverse Movement

Forced transverse movement of the jaw is usually accompanied by jaw opening in the sagittal plane to test muscles for hypertonicity and their normal ability to lengthen. It is necessary when testing forced jaw movements to determine whether the patient has a systemic ligament stretch reaction by testing other joints of the body for the condition (see Volume I). If present, it should be corrected first or it will be unknown during these testing procedures whether stretching the masticatory muscle or the ligaments is responsible for weakening of the indicator muscle.

Forcing the jaw into various positions is done by the physician or, preferably, a support person while the physician tests an indicator muscle. While passive jaw positioning is more desirable, sometimes testing can be done with the patient actively placing the jaw into the forced position. In the absence of a support person, the patient can be directed to hold the jaw in various positions for testing. If this is done, it must be determined that the patient does not demonstrate a positive therapy localization where he grasps the mandible.

When the jaw is opened and stretched to the side, the ipsilateral internal pterygoid and contralateral posterior fibers of the temporalis are stretched. This test is done with the patient first bilaterally therapy localizing to the temporomandibular joint



14—31. Posterior view of internal pterygoid muscle.



14—32. Intraoral contact to the internal pterygoid muscle for apparent neuromuscular spindle cell manipulation (see also figure 14—2).

while his jaw is maneuvered into the stretched position. If an indicator muscle weakens, the test is repeated with therapy localization to only one temporomandibular joint. If the mandible is opened and stretched to the right with right therapy localization and an indicator muscle weakens but does not with left TMJ therapy localization, the right internal pterygoid probably has a trigger point or requires fascial release technique. Under the same circumstances of jaw opening and stretching to the right, but with the positive therapy localization to the left TMJ and negative to the right, the posterior fibers of the temporalis are probably involved. It is possible for the middle or anterior fibers of the left temporalis to be malfunctioning, but that is not usual. In either case, the internal pterygoid or temporalis should be further evaluated for trigger points or fascial release technique. In the absence of referred pain from a trigger point in the muscle, the fascial release technique is applied as previously described.



14—33. Neuromuscular spindle cell treatment for hypertonicity of the posterior fibers of the temporalis muscle.

SEQUENTIAL MANDIBULAR MOVEMENTS WITH TL

As previously mentioned, testing and correcting the muscles of mastication with movement in the sagittal plane correct a large percentage of dysfunction. If there still appears to be dysfunction, testing and correcting transverse plane movement — both actively moved and passively stretched — correct another large percentage of the remaining problems. As we progress to the last and most integrated mandibular motion testing procedure, the answer will be found to most of whatever dysfunction is left — the more problematic interactions within the muscles of mastication. Usually if there is still a problem after this stage, it is not in the interaction of the muscles of mastication. Some other factor of integration within the total stomatognathic system or dysfunction remote in the body is probably interacting with the stomatognathic system. Examination of mandibular movement dysfunction which integrates with another division of the stomatognathic system or with the general body is discussed after jaw movement in the transverse and sagittal planes.

Evaluating a patient with movement in one plane and then another prior to testing an indicator muscle is indicated only when the previously described

testing procedures have been done and corrections completed, yet there is still evidence of improperly functioning masticatory muscles. This evaluation delineates muscle dysfunction responsible for a reactive muscle in a different sequence from that done in sagittal and transverse movement.

Two mandibular movements are sequentially made while the patient bilaterally therapy localizes to the temporomandibular joints. Immediately after the second movement is completed, but not into a stretched position, the examiner tests for a weakened indicator muscle. The first movement should be strong enough to make the muscles forcefully contract. The examiner must have an excellent knowledge of action of the muscles of mastication. It is necessary to know which muscles are active in the first and then the second mandibular motion. In this type of evaluation, the muscle needing treatment will be among the group that first activated mandibular motion, but the dysfunctioning muscle is among the muscles of the second action. This is apparently due to the first muscle sending inappropriate afferent impulses, causing the second muscle to react improperly. If the mandible is moved to the right and then opened, the primary activity is of the right posterior temporalis and the left internal and external pterygoid muscles. The secondary activity of jaw

AK Examination and Treatment of the TMJ and Muscles of Mastication

opening is of the bilateral external pterygoid muscles and anterior bellies of the digastric. If there is weakening of an indicator muscle following this sequential activity, further evaluate with therapy localization to only one temporomandibular joint. If therapy localization is positive to the right temporomandibular joint and negative to the left, evidence is that the posterior fibers of the temporalis muscle are hypertonic on the right. The posterior fibers of the temporalis are evaluated by therapy localization and palpation for an apparent neuromuscular spindle cell dysfunction. Treatment is to set down the spindle cell; it is accomplished by two-point manipulation toward the center of the spindle cell.

This type of examination for reactivity of the muscles of mastication helps to more specifically activate various muscles in sequence to aid in delineating a problem observed during the act of mastication. Sequential testing can be done with any movements the examiner can conceive. Some of the appropriate sequences and the muscles associated in prime movement are: (a) first, jaw opening, which activates the bilateral external pterygoid and anterior bellies of the digastric muscles and, second, lateral mandibular movement, activating the ipsilateral posterior fibers of the temporalis and contralateral internal and external pterygoid; (b) first, protrusion, activating the bilateral external pterygoid but not the anterior bellies of the digastric and, second, retrusion, activating the posterior fibers of the temporalis. Additional movements requiring the physician to discern which muscles are involved are protrusion to

lateral movement, retrusion to lateral movement, retrusion to open, etc.

This type of examination is indicated when the previous TMJ testing procedures are negative and there is no evidence of cranial faults, neurologic tooth, hyoid muscle imbalance, severe malocclusion, or overclosure; yet when the patient chews an almond or other low allergenic substance while bilaterally therapy localizing to the temporomandibular joints, an indicator muscle weakens. Chewing requires a complex interaction of the muscles during incising, movement of the food bolus, and trituration. Observing when the patient tests positive during various aspects of chewing may give the physician clues as to the muscles involved on a reactive muscle basis. For example, if the positive finding follows incising, the posterior temporalis and external pterygoid should be further evaluated because of their role in protrusion, necessary for incising. If positive during trituration, the muscles responsible for lateral movement should be evaluated in conjunction with the jaw-closer muscles. Evaluation is then done sequentially in both directions, that is, activating the lateral movement first and then the closing movement and, second, evaluating the closing movement and then the lateral movement.

Most muscle dysfunction responsible for reactive muscles will be found in the simpler testing procedures described earlier. It is the body language of dysfunction when other testing procedures have been found negative that brings the physician to these testing procedures.

MANDIBULAR MOVEMENT PLUS REMOTE FACTORS

Head and Neck Flexion or Extension

At the beginning of the discussion on evaluation of the muscles of mastication, it was stated that the supine patient should have his head and neck in a neutral position, not directed toward flexion or extension. It was observed relatively early in applied kinesiology evaluation of the temporomandibular joint that head and neck position could influence test results. When Goodheart first evaluated the muscles of mastication with the jaw moving in the sagittal plane, he observed that most involvements were of the jaw-closing muscles. As he taught these procedures to various dentists, they experienced more positive cases of the jaw-opening muscles. The answer to this dichotomy was found to be that on the typical chiropractic table the head and neck are held

in a flexed position by the headpiece, while in the typical dental chair the head and neck are directed more toward extension to facilitate jaw opening for dental procedures. It appears that the optimum testing position for the muscles of mastication is neutral. Additional testing in the sagittal or transverse plane can be done with the patient first flexing and then extending his neck and head. This testing parameter change brings other muscles of the stomatognathic system's closed kinematic chain into the picture. If the muscles of mastication test negative on TMJ testing procedures when the head and neck are in the neutral position, but positive when the neck and head are either flexed or extended, the other muscles of the closed kinematic chain should be evaluated. These include the

postural muscles — the sternocleidomastoid, upper trapezius, and deep spinal muscles — as well as the hyoid muscle complex. It will usually be found that there is dysfunction within these groups of muscles; when corrected, temporomandibular joint testing will be negative in the neutral, flexed, or extended neck and head position.

Another factor which can influence the temporomandibular joint during different neck and head positions is cervical subluxations or fixations. These, in turn, can influence the sternocleidomastoid, upper trapezius, and deep spinal muscles. There is often an interaction between cervical spine dysfunction and the muscles which support it. When movement of the head and cervical spine causes a positive TMJ, the usual spinal evaluation discussed in Volume I should routinely be done and corrections made. Other factors which may influence the muscle test when the patient changes head and neck position are disturbance in the rocker motion of the upper cervicals and occiput, and the possible need for PRYT technique.

Swallowing

Any or all of the TMJ testing procedures can be combined with swallowing to more thoroughly evaluate the stomatognathic system's closed kinematic chain of muscles. If a TMJ testing procedure is positive when the patient swallows before, during, or after the mandibular movement but is not positive without the swallow, there is evidence that the muscles of the hyoid complex are imbalanced. Proceed with the testing procedures described for the hyoid muscles in Chapter 13. After correction is accomplished there should no longer be a weakening of an indicator muscle during the TMJ test plus a swallow. If no disturbance is found in the hyoid muscles, the patient should be evaluated for orofacial imbalance as described in Chapter 15 on "Myofunctional Therapy." Cranial faults and malocclusion are also possibilities because of the intercuspation that takes place during swallowing.

Phonation

The temporomandibular joint and muscles of mastication tests can be combined with phonation similar to swallowing, which again tests the integration of the hyoid and orofacial muscles with the muscles of mastication. When a test is positive with phonation added to the TMJ testing procedures but not in its absence, the hyoid and orofacial muscles should be evaluated the same as discussed with swallowing.

There is some possibility that the patient could have improper balance between right and left brain

activity, causing the positive indication with phonation. In this case the patient will weaken when talking, whether or not the temporomandibular joint is being tested coincidentally.

Weight Bearing

All the previously described tests for temporomandibular joint function can be evaluated in a weight-bearing position, with the patient either seated or standing. Usually if there is a weight-bearing involvement it will readily be determined on the first weight-bearing test of jaw movement in the sagittal plane. If the TMJ is found positive in a weight-bearing position but not in a supine one, the total weight-bearing mechanism of the body should be evaluated. Testing in various positions helps locate the source of disturbance. For example, if the positive finding is present when standing but not sitting, the feet and knees are probable areas of disturbance. If it is positive also in the sitting position, the pelvis and spinal column are probably the cause. In any event, the weight-bearing mechanism should be evaluated as discussed in Volumes I and IV.

Regardless of head position or postural status, the engram controlling the muscles of mastication should adequately adapt to the postural change. McLean et al.³⁶ have evaluated the control of mandibular movement into centric occlusion with the subjects' position varied from standing to supine in increments of 30° by use of an electric tilt table. They found that the voluntary contractions of the jaw-elevating musculature were guided into almost identical registrations of centric occlusions, regardless of the subjects' position. When temporomandibular joint tests are negative in one position and positive in another, the additional parameters added to the test in the second position should be evaluated to find the cause of disturbance in the temporomandibular joints. The problem is usually not in the TMJ and its associated musculature in this situation.

Chewing

Possibly the maximum challenge to the temporomandibular joint and its associated muscles is the act of chewing. Not only does this action test the integration of the muscles of mastication, it also involves the occlusion, periodontal ligament receptors, cranial primary respiratory mechanism, all of the muscles of the closed kinematic chain of the stomatognathic system, and — if tested in various postural positions — the integration of the stomatognathic system with the rest of the body. Testing an individual while he is chewing is indicated only after other testing procedures have been done and the indicated corrections made. Simply have the patient

STRONG MUSCLE IN CLEAR

NO TL

RELAXED CENTRIC OCCLUSION

NEUROLOGIC TOOTH

MALOCCLUSION

DIMINISHED VERTICAL DIMENSION

CLEAR ALL OTHER FACTORS BEFORE COMING TO THIS CONCLUSION

MALOCCLUSION

CRANIAL FAULTS

IMBALANCE OF MASTICATORY MUSCLES

NEUROLOGIC TOOTH

PANTOTHENIC ACID

CHELATED IRON

HYPERTONIC CLOSING MUSCLES

CRANIAL FAULTS

TRIGGER POINTS

FASCIAL RELEASE

JAW MOVEMENT

JAW FORCED OPEN

FORCED CENTRIC OCCLUSION

NO TL TO TMJ

NO MOVEMENT

NON-FORCED MOVEMENT

SAGITTAL MOVEMENT STOPPED IN:

TRANSVERSE MOVEMENT STOPPED:

SEQUENTIAL MOVEMENTS FOR REACTIVE MUSCLE:

INDICATOR STRONG AFTER FIRST MOVEMENT BUT NOT AFTER SECOND

JOINT OR DISC PATHOLOGY

DISC DYSFUNCTION

BONE PATHOLOGY

OPEN EXT. PTERYGOID ANT. DIGASTRIC

CLOSED MASSETER INT. PTERYGOID TEMPORALIS

RIGHT POST. FIBERS TEMPORALIS

LEFT INT. AND EXT. PTERYGOID

ABDUCTION TO OPEN

ABDUCTION TO CLOSED

OPEN TO ABDUCTION

CLOSED TO ABDUCTION

PROTRUSION TO RETRUSION

RETRUSION TO PROTRUSION

OTHER CONTRIBUTIONS AS INDICATED DURING CHEWING

DETERMINE SIDE BY ONE-HAND TL

TL PLUS MOVEMENT AND OTHER FACTOR

HEAD AND NECK FLEXION AND EXTENSION

STERNOCLEIDOMASTOID

UPPER TRAPEZIUS

DEEP FLEXORS

DEEP EXTENSORS

CERVICAL SPINE

PRYT. TECHNIQUE

ROCKER MOTION

SWALLOWING OR PHONATION

HYOID MUSCLES

OROFACIAL MUSCLES

WEIGHT BEARING

ANY FACTOR IN THE WEIGHT-BEARING COMPLEX

CHEWING

THE ULTIMATE CHALLENGE TO THE TOTAL COMPLEX OF THE STOMATOGNATHIC SYSTEM

chew various types of food while bilaterally therapy localizing to the temporomandibular joints, and test an indicator muscle. When this test is positive, further testing of the stomatognathic system is indicated.

For thorough evaluation of the masticatory system various types of food should be chewed, as the muscles are activated differently with hard food compared with soft, more viscous food. During the chewing of hard nuts the electromyographic pattern is steep, shows high amplitude, and is of short duration. Chewing soft, viscous food shows a longer-lasting, waxing and waning type of electromyographic activity.³⁰ Incisal activity, such as biting an apple, should also be included in a thorough evaluation.

When chewing is included in the masticatory evaluation, the type of food should be considered. It is well-known in applied kinesiology that various nutritional and other substances, when placed in the mouth, can either make a previously strong muscle weak or a weak one strong. If a person is given a substance to chew, it must be known that the substance itself does not cause an indicator muscle to change strength. Items usually applicable for

testing mastication are raw almonds with no additives, natural cheeses, and warmed parafin. These substances can be evaluated by having the patient simply suck on them with no masticatory activity while an indicator muscle is tested for weakening.

This discussion of evaluation of the muscles of mastication and temporomandibular joint function may seem rather long and extensive. In clinical use evaluation is very quickly accomplished, along with the necessary corrections indicated by the examination. The key factor for the doctor's efficiency in its use is to be very familiar with the anatomy and action of the muscles of mastication, the integration of the stomatognathic system, and the stomatognathic system's integration with total body function. With this knowledge the primary disturbance which may be causing many random health problems can be found and corrected by simply working with the body's own abilities as a self-correcting, self-maintaining mechanism. The doctor knowledgeable in these examination and treatment procedures will prevent many iatrogenic problems which may be created by others who treat symptoms and effects rather than finding the basic underlying cause.

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Chapter 15

Myofunctional Therapy

Daniel Garliner
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Introduction

The term “myofunctional therapy” was coined by B. E. Lischer,⁵⁵ who — along with others in the early 1900s — began considering involvement of the orofacial muscles with dental irregularities. Among the very early proponents of evaluation and therapy to the orofacial muscles was Rogers, who first published exercises for the development of the facial muscles in 1918⁷⁷ and continued to publish additional information on several occasions.^{78, 79, 80} Most of his discussion was in reference to the masseter, temporalis, and other muscles of mastication, as well as to the facial muscles. Although he mentioned the tongue, there is no description of training it in swallowing as accomplished in modern myofunctional therapy. In 1950 Rogers published “A Restatement of the Myofunctional Concept in Orthodontics.”⁸¹ In referring to the orthodontic profession’s use of myofunctional therapy he stated, “. . . thus far I fear that I have failed to make a very deep and lasting impression upon many of our profession. It is because of this that I appear once more before you to make one more endeavor to stress the value of myofunctional treatment, not alone because of the satisfaction its intelligent application can bring to the orthodontist, but to the many benefits it can bring to the growing child.” These comments to the American Association of Orthodontists aptly reflect the minimal interest the profession had shown in myofunctional therapy at that point in time.

The profession’s interest in myofunctional therapy was spearheaded by Straub in the early 1960s. His

first article in 1951⁹¹ on what was then called the perverted swallowing habit did not gain much attention. A series of articles published in 1960-61-62, Parts I, II, and III,^{92, 93, 94} on the abnormal swallowing habit — along with his lectures throughout the country — brought a new interest to this subject.

In modern myofunctional therapy, considerable attention is given to the swallowing mechanism, with emphasis on the tongue’s action. There is conflicting research concerning almost every aspect of myofunctional therapy, ranging from its etiology to its effect on dentition¹⁰¹ to therapy effectiveness.¹⁸ An example of conflicting thoughts is apparent in the statement, “Swallowing is a complex reflex action and is not likely to be re-trained successfully and permanently.”⁴⁰ This is in complete disagreement with routine clinical evidence as observed by the Payne technique, Mandibular Kinesiograph, force scale, and Myoscanner, discussed later in this chapter.

The procedures used in myofunctional therapy have developed in spite of much controversy, and today there are organizations certifying myofunctional therapists who are concerned with further research in the field. Much of the controversy developed as a result of unqualified personnel attempting myofunctional therapy.^{29, 73} Failure to obtain positive results was blamed on myofunctional therapy, rather than on the individual providing the training. This is similar to the problem applied kinesiology has experienced. It seems that anyone doing muscle tests is classified as “an applied

Myofunctional Therapy

kinesiologist,” even though the training and background necessary to provide effective examination and therapy are lacking; consequently, applied kinesiology is blamed for inappropriate diagnosis and ineffective therapy when, in reality, the blame should be placed on the practitioner.

Those who recognize the term “myofunctional therapy” generally think it refers to correction of anterior tongue thrust, a major muscular dysfunction of the orofacial complex which is recognized and treated. Current myofunctional therapy gives considerable attention to correct tongue function during swallowing, but it has a broader scope of application to all muscles in the orofacial complex, and to correction of various adverse habit patterns influencing this area. As with other muscles of the stomatognathic system, the orofacial muscles must be organized with the balance of the entire body; they are important in establishing the physiologic centric relation.⁴⁷

In addition to the tongue's function during swallowing, this chapter considers the etiology of abnormal swallowing patterns and correction of adverse habit patterns, such as thumb-sucking, lip- and cheek-biting and sucking, and lip-licking. Sucking and biting on objects such as blankets, locks of hair, pipes, toothpicks, and match sticks are habits which must be considered when dealing with the stomatognathic system because they often influence swallowing or affect the occlusion. The habit may adversely influence the occlusion, or it may be a subconscious effort to balance malocclusion or prevent a sensitive prematurity from making contact. Mouth breathing, if present, must be investigated and the condition corrected to normal nasal respiration because of its influence on swallowing and orofacial muscular balance.

One of the major activities of a myofunctional therapist is to re-train the orofacial muscles in order to correct an improper swallowing habit and permanently establish the correct procedure. A professional trained in these procedures, therefore, can establish proper habit patterns in other areas which do not directly deal with orofacial muscle balance, but do influence it. There are many habits which fit this pattern. Especially detrimental is propping the chin or some other cranial area on the hand while studying, reading, watching television, etc. These stresses to the jaw and cranium often cause recurrent cranial faults and temporomandibular joint dysfunction, even after excellent therapy is provided for correction. In the same vein, improper sleeping positions can re-create problems.^{87, 88, 89} These habits, as well as others, are maintained on a semi-subconscious level; their correction requires the

expertise of an individual familiar with the procedures and psychology of adverse habit correction.

Many professionals providing treatment for various types of conditions need to be familiar with the concepts of myofunctional therapy.^{20, 21} They should be trained in its therapeutic procedures or have a qualified myofunctional therapist available for patient referral. Physicians interested in the general subject matter of this text should especially be cognizant of myofunctional therapy. Muscular imbalances in the orofacial complex are important in correcting or causing cranial faults, and they directly influence the development and function of the occlusion.

A percentage of patients with symptoms relating to the temporomandibular joint requires myofunctional therapy, and many individuals who are seen by a myofunctional therapist have symptoms relating to the temporomandibular joint.⁸⁶ It is important that professionals taking care of these patients recognize additional involvement apart from their field and make appropriate referrals, when necessary. For example, a myofunctional therapist may initially see a patient for speech therapy, but a referral may be necessary for malocclusion,²⁷ temporomandibular joint dysfunction, or other involvement in the total stomatognathic system. Likewise, a dentist or a chiropractor may see an individual with headaches and/or facial pain as a result of temporomandibular joint disturbance who requires additional treatment from a myofunctional therapist. Of particular interest to the applied kinesiologist is the influence orofacial muscle imbalance has on the dental occlusion.^{24, 26} Many resistant cases with cranial faults are due to this type of imbalance. As with other areas of interprofessional communication, it is obvious that there must be increased communication between the treating physician and the myofunctional therapist.²³

Earlier in this text there was discussion of how facial muscle imbalance can create or perpetuate cranial faults. It has also been pointed out how imbalance of the masticatory muscles can adversely affect the occlusion and temporomandibular joint function. Sometimes orofacial muscle imbalance is due to local trauma to a muscle; this can usually be easily corrected with techniques used in AK, such as treatment to the muscle proprioceptors, fascial release, or spray and stretch. Probably of greatest importance in the applied kinesiology treatment of the orofacial muscles is the correction of cranial faults, returning normal nerve supply by removing the peripheral nerve entrapment created by the fault.

Much imbalance in the orofacial complex is created by improper swallowing patterns and adverse habits. This type of etiology can cause a muscle or muscle group(s) to develop improperly, with disuse

atrophy or hypertrophy of the muscle(s) following. When a habit pattern is present, such as improper swallowing, a new and permanent engram must be developed; this takes the persistence and talent of a myofunctional therapist to establish. If there is hypertrophy or lack of muscle mass from disuse atrophy, specific exercise programs are required to return or obtain normal balance. The applied kinesiologist unaware of the need for myofunctional therapy in these conditions will fail to obtain permanent results with his usual treatment or, at best, will not bring this patient to an optimum health level. In 1977 Leaf⁵¹ reported improved cranial function after having an individual with an atypical swallow correctly swallow five times on inspiration. His findings concur with ours, showing evidence that when the swallowing mechanism and other aspects of the stomatognathic system are balanced, the cranial primary respiratory mechanism improves.

After diagnosis of orofacial muscle dysfunction, the procedures in myofunctional therapy are primarily educational and motivational. The therapist must be an individual with a high level of interest in and good rapport with the patient. A considerable amount of psychological interaction is necessary with the patient; this ability can be acquired, but it is usually best provided by an individual with a natural, almost innate ability for interpersonal communication with the general population.

The purpose of this chapter is not to teach myofunctional therapy; it is to acquaint the physician with its procedures and how to determine its need. A physician may wish to make an in-depth study of myofunctional therapy in classes designed to provide training and practical application of the procedures. He may choose to provide the myofunctional therapy himself once training is completed, or use this knowledge to monitor a therapist working either in his office or on a referral basis. Myofunctional therapy is usually provided by a speech consultant or a dental hygienist or assistant because of his or her prior training in the anatomy and physiology of this portion of the stomatognathic system.

Speech consultants have an advantage in the training necessary for a myofunctional therapist

because they have been taught to change the habit patterns of tongue placement during speech. Although familiar with the anatomy and physiology of the area relating to speech, they are usually handicapped by a lack of knowledge in dental occlusion, terminology, and function of the cranial primary respiratory mechanism. Dental hygienists and assistants are familiar with the anatomy and physiology of the stomatognathic area and knowledgeable in dental occlusion and TMJ function, but they are handicapped by not having the training for habit change which the speech consultant has.

As mentioned, myofunctional therapy is a specialty area of habit correction and motivation. Excellence in the procedures requires training, dedication, and practice. Although I recognize the importance of myofunctional therapy, I do not apply it in my practice; rather, I observe the need for it for referral purposes. Some of the muscular imbalances observed can be corrected with applied kinesiology procedures; these are treated within the office. The conditions of improper development, poor habit patterns, and disuse atrophy should be referred to a myofunctional therapist for in-depth training and motivation.

This chapter is greatly enhanced by its co-author, Professor Daniel Garliner, President of the Institute for Myofunctional Therapy which he founded in Coral Gables, Florida.⁴³ A speech consultant and myofunctional therapist, he has a wealth of background knowledge from having practiced myofunctional therapy for sixteen years. He is also a Visiting Assistant Professor, Department of Orthodontics and Prosthodontics, Howard University Dental School, Washinton, D.C.; Clinical Member, T.M.J Clinic, New York Eye and Ear Infirmary, Department of Otolaryngology, New York, New York; and Visiting Lecturer at many universities throughout the United States and Europe. My appreciation is extended to Professor Garliner for his contribution to this text and to humanity for improved understanding of function and therapeutic techniques.

David S. Walther, D.C.

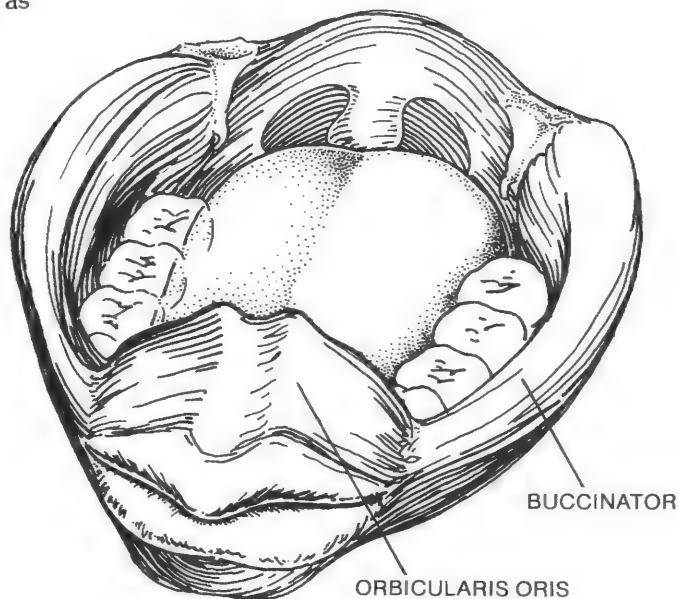
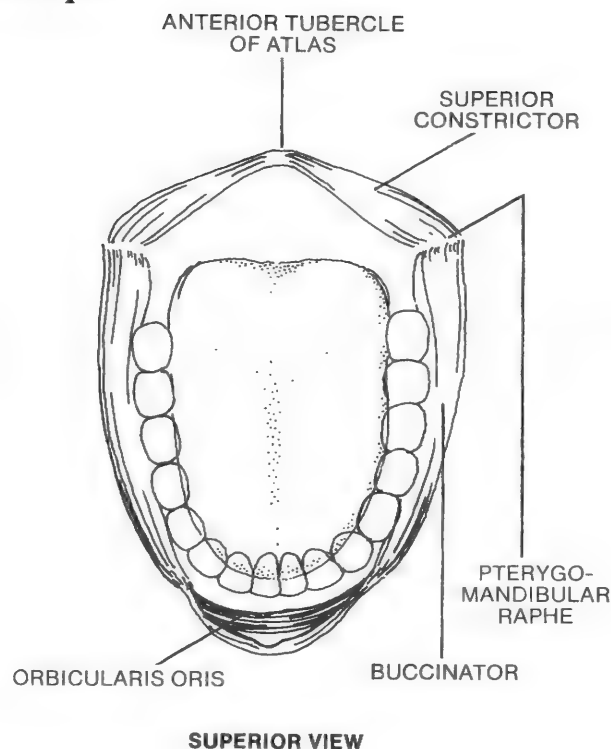
Muscle Envelope

There are four groups of muscles which influence dentition — the muscles of posture, deglutition, mastication, and facial expression.¹¹ They also influence the cranial primary respiratory mechanism, as does dentition. This makes these muscle groups of significant interest to the applied kinesiologist because they are applicable to AK examination and treatment. The muscles of deglutition, mastication, and facial expression generally require indirect testing because individual manual muscle tests are not applicable. Individual testing procedures and treatment are discussed in the appropriate sections of this text, such as in the chapters on TMJ Function (Chapter 11), AK Examination of Organization within the Stomatognathic System (Chapter 16), Skull Muscles (Chapter 4), and Hyoid Muscles (Chapter 13). In this chapter we deal with how the orofacial muscles influence dentition and special methods for testing some of them.

The muscles of concern as an envelope to the dentition are those surrounding the dental arch. Externally they are the orbicularis oris and the muscles inserting into it, including the buccinator; internally they are the muscles making up the tongue. These muscles result in an envelope that surrounds the dental arches. Normally there is equilibrium in the muscles at rest, resulting in equal pressure on both the facial and lingual sides of the teeth. The balance of these muscles and their influence on the dentition have become known as "an equilibrium theory of tooth position."

The normal resting position provides a freeway space between the teeth which is programmed for an individual. The tongue rests between the dental arches, with or without contact to the palate. There is minimal lateral or anterior force on the dental arches, or pressure against the palate. Externally, the orbicularis oris muscle is influenced by the insertion of the levator labii superioris, levator labii superioris alaeque nasi, levator anguli oris, zygomaticus major and minor, risorius, depressor labii inferioris, depressor anguli oris, and mentalis, along with the buccinator.

The outer portion of the muscular envelope and the tongue as the inner portion, along with the forces inherent in the arches, must equalize each other in function. This causes a dissemination of force so no moving pressures are exerted on the teeth.⁷² Constant force on the



15—1. Muscle envelope of the teeth.

dental arch will change its shape and the position of the teeth. The constant nature of the force is a most important factor in influencing bone growth and tooth position. Note the grooves on the internal aspect of the skull formed by the constant pressure of blood vessels. The same type of constant pressure is present when there is muscular imbalance. The often repeated comment, "When bones and muscle fight, muscle never loses," is quite applicable here. Muscle function influences bone shape and growth, and also the type of suture.^{11, 57, 58, 99}

The influence of muscle on bone is dramatically demonstrated in a study by Harvold,³⁹ where a piece of plastic was anchored to the palate between the right and left maxillary molars of monkeys. This changed the tongue's function, disturbing the muscular envelope and causing additional pressure from within. After several months, the animals developed an open bite and multiple diastemata.

In another study, a wedge was excised from the monkey's tongue, putting less pressure on the dentition by the internal aspect of the envelope. "After four to six months, the response expressed itself as crowding of the teeth and a deeper bite."³⁹ In a similar study, thirty rats were divided into three equal groups; one was the control group, another was subjected to partial glossectomy, and the third group had a total glossectomy. After three months, the upper and lower jaws of the rats in the third group were smaller than those in the control rats. There is no discussion regarding the effects on the rats in the second group subjected to partial glossectomy in this Italian study.⁶¹

This discussion centers primarily on how imbalance or dysfunction of the orofacial muscles can adversely influence the dentition. It should be recognized that there has been considerable controversy as to whether the muscles influence the dentition (function determining form), or whether the dentition influences muscle function (form determining function). There are proponents of both viewpoints, with some taking a very strong stand on one side of the issue. As with so many other controversies in physiology, a study of both viewpoints indicates that neither has the complete answer; some cases of malfunction are predominantly on one side and some on the other side, with still others being a combination of both.^{27, 37}

Form dictating function is exemplified by a genetically determined high, narrow palate which does not provide adequate room for the tongue to function. On the other hand, an apparently similar condition can be an example of function determining form when the tongue does not rise into the palate with sufficient force during swallowing; this results in

a failure of action of the inner aspect of the muscular envelope. Here the tongue fails to provide the counteracting force to the circumoral muscles. The answer to determining whether function determines form or form determines function is differential diagnosis, indicated later in this chapter, with muscle testing instruments and swallowing evaluation.

For the position of a tooth to change, there must be a constant force applied to override the effectiveness of the periodontal transmission mechanism, which is the suspension of the tooth by the periodontal fibers. The amount of force required varies considerably with the type of root, health of the periodontal tissues, and direction of force applied.¹⁶

To understand the intricacy of the balance of the orofacial muscles, the amount of pressure required to move teeth in their periodontal transmission mechanism must be understood. Lear et al.⁵² studied both time and force applied to teeth and noted an inverse ratio. When the force was applied for a shorter period, there was less tooth movement. Even so, small forces of only 25 msec duration were capable of tooth displacement. In analysis they state, "This study indicates that small forces are capable of displacing pre-molars. The displacement can be initiated by constraints similar in magnitude and direction to those that are received by pre-molars from the tongue and cheeks when at rest."

Considerable pioneering research has been done at the University of Nebraska regarding the equilibrium theory of tooth position. Much of this has been reported by Weinstein et al.¹⁰² A tooth is in equilibrium when the resultant of all forces on the tooth equals 0. This must take into consideration the force applied by the muscular envelope, adjacent teeth, forces from occlusion, bone, and the periodontal tissues, as well as the force of eruption and mastication of a food bolus. Foreign objects, such as a thumb, finger, appliance, etc., must also be considered.

The first effort was to determine the amount of force applied to the dentition by the muscular envelope. By use of a transducer, the force of the muscles on the various aspects of the dentition was determined with different types of activity, such as tongue and lip movement, sucking, reading a short paragraph, etc. In order to test how these muscular forces influence the dentition, a 2 mm gold inlay was attached to the first bicuspid. Unmodified first bicuspid of each subject were used as controls. In one study,⁵ both the modified and control teeth were relieved of all proximal and occlusal contacts to ensure complete freedom of tooth movement. It should be noted that these investigations were done

Myofunctional Therapy

on individuals who had previously been selected for extraction of four permanent first bicuspid as part of their orthodontic treatment; thus the modifications to the teeth were not damaging to the subjects' final dentition. Movement of both the control and modified teeth was measured weekly by a dial indicator, accurate to .001 inch. In general there was minimal movement of the control teeth. The significant movement of the modified teeth was up to .033 inch, which is close to half the amount of the 2 mm inlay modification (1 mm = .03937 inch). The modified teeth moved in the appropriate direction to equalize the force applied by the muscles as a result of the modification. An exception to this was when one of the lingually modified teeth did not come into contact with the tongue, apparently because of the individual's habit pattern of tongue position. In one case when the control tooth moved significantly, it was suspected to be due to exfoliation of the adjacent second deciduous molar. Minimal movement of some control teeth was probably due to the relief of the occlusal and proximal contacts which, of course, was a part of the total equilibrium of forces on these teeth.

A follow-up study to Attaway's was done by O'Meara,⁶³ where a similar 2 mm modification was made to a tooth with a gold onlay. In this study there was no modification to the occlusal or proximal surfaces of the teeth to determine if the tooth would still move from imbalanced muscular forces when the teeth were in their existing functional environment. There was less movement of the unmodified control teeth in this group than in the original study, and there was less overall movement of the modified teeth. In two cases, the tooth moved toward the direction of modification, which appears to be opposed to the hypothesis of muscular forces moving teeth. The analysis of these variances was that pressure was exerted during mastication by the food bolus, placing force on the onlay to move the tooth in the direction opposite that expected. This hypothesis was supported by modifying the onlay of one tooth which was moving in the opposite direction from that expected. After modification the food bolus did not contact the onlay with as much force; the tooth changed its direction of movement to that expected by the equilibrium theory.

The ability of the teeth to move from change in muscular force but without any change to the proximal or occlusal surfaces is important in applied kinesiology. When cranial faults and muscular imbalance are corrected, there may be a change in the occlusion. It is important that the teeth be able to move by remodeling forces to adapt to these changes. As has been discussed, it is sometimes

important for a dentist to equilibrate the occlusion to the new environment when massive changes are made. When changes of the muscle function and cranial mechanism are made more slowly, it is important that the dentition be capable of adapting to its new environment.

In a subsequent study, Weinstein¹⁰³ correlated the findings of numerous University of Nebraska investigators to better understand the minimal forces required for tooth movement. The elastic quality and force of the cheek over the pre-molar region was related with the 2 mm modification of the tooth. It was determined that muscle forces of as low a value as 1.68 gm above the resting force will move teeth, if the force is applied over a sufficient length of time. Other studies revealed that when the onlay is removed, the tooth will return to its original position.³⁸ The tooth's position depends on its total equilibrium, which includes the forces of occlusion. It has also been shown that masticatory forces can produce a buccal tipping tendency on the lower pre-molars, changing the equilibrium and contributing to the tooth's position.¹²

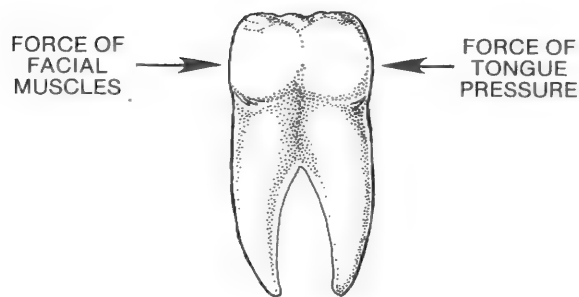
The dimension of time appears to be an important factor in the equilibrium theory of tooth position. In the studies noted above, the tooth moved progressively through the time span studied. In most cases the tooth was measured on a weekly basis for eight weeks or longer. Kydd et al.⁴⁸ found that subjects with an anterior open bite had a statistically significant increase in duration of the lip and tongue pressures applied to the anterior teeth during swallowing, as compared with a control group. In this study tongue thrusters who had been treated orthodontically for an open bite and had relapsed exerted more than twice as much pressure anteriorly, and only two-thirds as much lip pressure as those whose orthodontic correction remained stable.

A study of the open bite by Wallen⁹⁷ used a transducer which could be positioned at various angles to evaluate the pressure of the tongue during swallowing. Subjects with normal occlusion and with open bite were contrasted. Those with open bite had approximately equal pressures in all planes, while those with normal occlusion had higher pressures in the vertical and intermediate planes than in the horizontal plane of space. This would seem to indicate that form dictates function in this type of open bite. The study must be questioned, however, because the dimension of time was not included in the pressures exerted in the various planes.

There are many additional studies which indicate that tooth mobility depends upon the forces applied to the tooth.^{16, 96} A study by Rogers⁸² separated groups of children with normal occlusions from those

with malocclusion. He found a greater presence of tongue thrust among those with malocclusion than among those with normal occlusion. Similar findings are evident when a child is evaluated for an open swallow. This is the type of swallow present in the typical tongue thrust, where the molars are not brought into intercuspation during swallowing. Rix⁷⁶ found in sixty-one children who swallowed with the teeth in occlusion that the dentition deviated from normal in 36%; in twenty-seven children who swallowed with the teeth separated, 81% had dentition that deviated from normal.

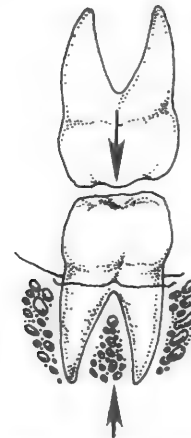
It becomes obvious that muscular balance of the dental envelope is very important in the formation of the dental arches, occlusion, and crooked teeth. We are not recommending that myofunctional therapy take the place of orthodontic treatment,²⁹ although Rogers^{80, 81} colorfully refers to the muscles as being "... living orthodontic appliances." It certainly does make sense that correcting factors which cause imbalance of the muscular envelope may prevent disturbances that would otherwise develop. Balancing muscle dysfunction helps obtain more rapid orthodontic correction and prevents relapse after the appliances and retainers have been removed. It also helps prevent collapse following proper oral surgery.²¹



15—2. Forces exerted on teeth by muscles. There is also force from the proximal teeth.

The forces on a tooth are not limited to those on the mesial and distal surfaces; also included is force directed axially toward the root and the crown.³⁴ The axial force toward the root is that of occlusion, which is very strong and is counteracted by the forces of eruption. These axial forces must equalize each other or there will be supereruption or intrusion of the teeth. There is also a certain amount of force equalization necessary regarding the contact point of the proximal surfaces of the teeth. It is well-known

FORCE OF OCCLUSION



FORCE OF ERUPTION

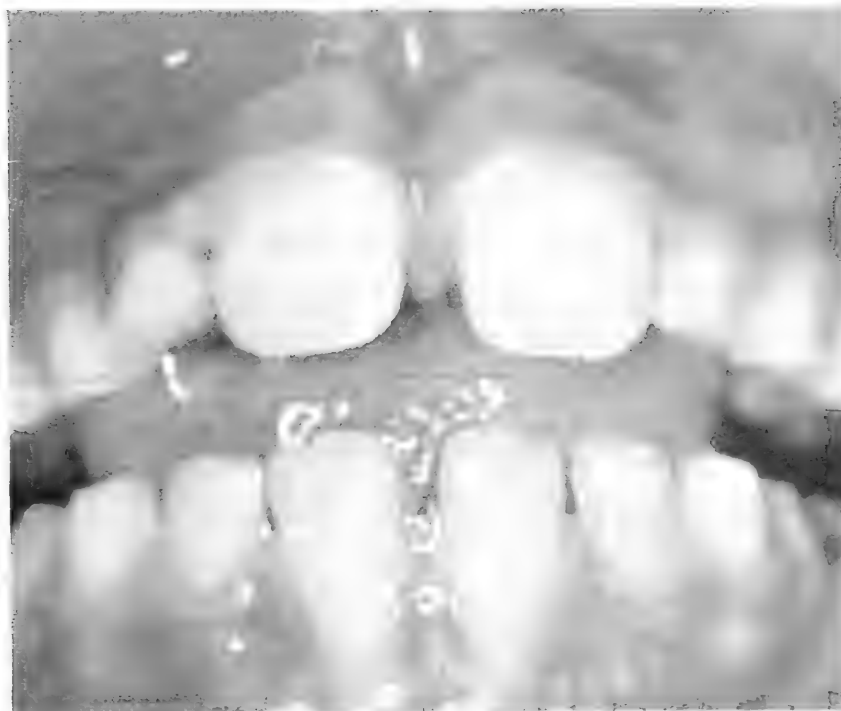
15—3. Axial forces applied to teeth.

that loss of a tooth allows an adjacent tooth to migrate into the vacant area. This is countered by replacement of the missing tooth with an artificial device, such as a fixed or removable bridge, or with a temporary acrylic space maintainer.

The axial forces are disturbed when there is a lateral tongue thrust between the teeth. This occurs when an individual inserts his tongue between the posterior teeth while swallowing. This provides a stop which may cause the teeth to fail to erupt to their proper potential; if they have already erupted to their maximum they may be intruded, causing an open bite. The importance of the forces during swallowing is emphasized by measurements which show twice as much force applied to the teeth during a day by swallowing than by chewing.⁴⁹

When there is an imbalance of forces on a tooth, the disturbed position may be minimal and require only routine muscle correction with AK procedures; myofunctional therapy, orthodontics, or equilibration may be necessary to return normal occlusion. In any event, dealing only with the occlusal problem and not correcting the muscular imbalance may be treating the effect rather than the basic cause of the condition.

The function of the inner and outer muscular envelopes must be such that there is a dissemination of force. If one muscle consistently presses harder against an area of the dental arch than its opposing force, the structure will yield to the stronger force.



15—4. *Typical appearance of tongue thrust.*

On the other hand, there can be normal pressures applied to the dental arch by muscle but failure of an opposing force; the structure will yield to the normal force. The opposing forces of the tongue and lips give us an example of both types. If there is anterior tongue thrust and normal lip function, the tongue may push the teeth anteriorly, causing an Angle Class II malocclusion. On the other hand, if the orbicularis oris is weak there will be nothing to restrain normal tongue action, and a similar structural change may develop.

The discussion to this point has been confined to the dysfunctioning orofacial muscles' effect on the natural dentition. Another type of problem from this dysfunction should be recognized. There are many individuals with numerous sets of "ill-fitting dentures" or partial prostheses which give constant trouble because of orofacial muscle imbalance.⁸⁶ Often the reason for loss of teeth is orofacial muscle imbalance, resulting in malocclusion and periodontal disease. While the replacement prosthesis is designed and equilibrated with great care, it may be the victim of the same orofacial muscle imbalance that caused the loss of the teeth in the first place. It is sometimes necessary for the prosthodontist to at least temporarily build into the prosthesis an open bite or other form which parallels the original malocclusion to

accommodate the improper orofacial muscle function. In some cases, myofunctional training can correct the muscular activity to accommodate a properly designed prosthesis. There may be limits to the myofunctional success because the age of an individual may produce a deeply seated engram of muscle function which cannot be changed.

In the typical individual with tongue thrust, the masticatory muscles fail to contract adequately during swallowing.^{76, 92} Lack of action of the masticatory muscles is even more prevalent when the orofacial imbalance results in a posterior open bite. It was pointed out earlier in this text that contraction of the muscles of mastication during swallowing is an important mechanism for activating the cranial primary respiratory mechanism. Not only does the cranium obtain activation, but a balancing

effect also takes place to correct cranial faults resulting from the various pressures applied to the cranium during a typically active day. The upper arch, made up of the maxillae, is pulled against the lower arch in the more stable mandible; this tends to balance the cranium when no malocclusion is present.

Mentioned earlier were the four groups of muscles which influence dentition — the muscles of posture, deglutition, mastication, and facial expression. As has been pointed out throughout this book and others on applied kinesiology, there is an interactivity among various muscle groups of the body; so it is with these groups.³⁴ Disturbance in any group will probably adversely affect the others. Here we are dealing primarily with the muscles of deglutition and facial expression, and secondarily with the muscles of mastication and posture. If the muscles of deglutition and facial expression dealt with in myofunctional therapy are imbalanced, an occlusal problem will probably develop which disturbs the balance in the closed kinematic chain discussed in Chapters 8 and 10. This could lead to equilibrium disturbance, and the postural muscles of the body could become imbalanced. The vicious circle has developed to potentially cause health problems throughout the body, and it may have resulted from something which seems as simple as improper tongue activity during swallowing.

Etiology of Orofacial Muscle Imbalance

Orofacial muscle imbalance can be considered as anything which disturbs the equalization of muscular forces on the dental arches. This definition includes the muscles of deglutition, which bring the supra- and infrahyoid muscles into the complex, and also the tongue. The muscles of mastication are included in the swallowing mechanism, and of course are more involved during mastication with force applied to the occlusal surface in various directions. It also includes the muscles of mastication in abnormal patterns, such as bruxism or bracing.

There are numerous etiologies for imbalance of the orofacial muscle complex. Sometimes the muscle

imbalance is primarily from structural distortion caused by improper developmental patterns, adverse habit patterns, or trauma causing an improper form (form determines function). Other times the same basic principle in all applied kinesiology relates to the orofacial muscle complex. When the muscular forces are balanced, structure functions correctly (function determines form); when they are out of balance, disturbed form results. Specifically relating to tooth position, the activity of the muscles must be balanced in order for the force applied to the teeth to be disseminated properly, thus maintaining the balanced position of the teeth and arches.

DEVIANT SWALLOWING PATTERNS

The most common condition treated by a myofunctional therapist is abnormal orofacial muscle activity during swallowing. As discussed in Chapter 10, a normal swallowing pattern is characterized by the tip of the tongue touching the incisive papilla, the middle of the tongue contacting the palate, and the back of the tongue dumping food into the oropharynx. Cinefluorographic studies by Jankelson et al.⁴⁴ reveal that the tongue has a very forceful piston-like thrust from anterior to posterior during swallowing. A deviant swallowing pattern is characterized by a change from the normal, with the tongue thrusting against the anterior dental arch or possibly thrusting laterally into the posterior dental arch. The first type recognized was that of anterior thrusting, simply called tongue thrust. The term has broadened

to include all forms of abnormal tongue movement, both during swallowing and when at rest.^{9, 25}

A specific swallowing pattern develops for each individual. Electromyography reveals that there are variations of swallowing patterns between individuals, but the pattern demonstrated in one person is consistent for him.⁴¹ The same study reveals that an individual's swallowing pattern does not change with postural change or with the type of bolus being swallowed. Myofunctional therapy is designed to retrain the consistently abnormal swallowing pattern to one which is balanced with the other muscles in the orofacial complex. In addition to teaching the correct pattern for swallowing, it is necessary to firmly establish a permanent habit pattern.

CAUSE OF TONGUE THRUST

There appear to be many causes of tongue thrust, and there is much controversy regarding its primary cause⁹ and its effect on the dentition.⁹⁵ Many younger children swallow with a tongue thrust which develops into a normal pattern without therapy. Hanson and Cohen³⁷ studied 178 children, finding that at four years and nine months of age 103 (57.9%) were tongue-thrusters. The children were re-evaluated without therapy at five years and eight months, six years and seven months, seven years and five months, and eight years and two months. During

that period of time, sixty-three (61.2%) spontaneously developed a normal swallow. Of the seventy-five (42.1%) who originally had a normal swallow, twenty-five (33.3%) developed tongue thrust by the age of eight years and two months. Some of the children were "transitional thrusters" who showed tongue thrust only during some of the five examinations that represented the total study. Another longitudinal study⁴⁵ concluded that there was no improvement of tongue thrust from maturation alone. The primary consideration of the physician or therapist in charge

Myofunctional Therapy

is to determine by examination procedures whether the tongue thrust continuously interferes with normal function. If so, therapy should be initiated to return normal function to the orofacial muscles to prevent further distortion of function. This is particularly important in children during their growth period.²²

It appears that there are many causes of a deviant swallowing pattern. It may be a pattern which develops in early infancy and continues indefinitely, to be corrected only by a training program with myofunctional therapy. A normal pattern may develop in infancy which is disturbed by some functional factor causing the development of tongue thrust. If this factor is corrected, the tongue thrust may return to normal without therapy. It is important to evaluate for probable etiology because it may have a bearing on the success of therapy. For example, it appears that some tongue thrusting develops as a result of mouth breathing created by airway restriction, such as that caused by allergies. If an allergy is not corrected and a child continues mouth breathing, the success of myofunctional therapy may be limited. It seems that most deviant swallowing results from improper orofacial muscle development in the infant, often due to bottle-feeding rather than natural breast-feeding.

Bottle-Feeding

Many have suggested that the development of tongue thrust is due to bottle-feeding. Straub⁹² related tongue thrust in 478 children to the habit pattern developed by the tongue during nursing. Only two out of this group were breast-fed, and in their case the mothers had a heavy free-flow of milk. It is suspected that an infant controls the overabundant flow of milk by thrusting the tongue forward. Garliner²⁵ studied 985 cases of deviant swallowing and found that only seventy-five (7.6%) were completely breast-fed. In questioning the mothers who completely breast-fed their children it was found that they had a very rapid, free-flowing milk supply. When the overall population of infants is considered, only a small percentage will be completely breast-fed. Even those who start out with total breast-feeding are often fed from a bottle as additional food items are introduced into the diet, such as orange juice, etc.

Several studies indicate that bottle-feeding is not the primary etiology of orofacial muscle dysfunction.^{1, 9, 10} An overview of the total problem indicates there are probably several reasons for the orofacial muscle dysfunction. As Straub,⁹¹ an early proponent of bottle-feeding as the etiology of abnormal swallowing points out, leaning habits, thumb- and finger-sucking, lip-biting, nail-biting, etc., can be the

etiology. In any event, there are many positive aspects to nursing an infant, and it should be encouraged for the psychological and nutritional effects, as well as for possible functional effects. As Picard⁶⁷ states, "If all newborn could organize, they quickly would engage an advertising concern which would in short time succeed in convincing the American mother of the nutritional value of the product of her mammary glands."

Applebaum³ cites many references regarding the physical advantages which accrue to both infant and mother. There is less infant mortality and morbidity, less respiratory disease, and a reduction in infectious diarrhea. Allergy and eczema are reduced, and there is a psychological advantage to the infant. The mother also derives benefits since there is less pelvic congestion, which decreases the frequency of post-partum rectal hemorrhoids and leg varicosities. The feminine figure returns to normal more rapidly, and there is also a psychological advantage to the properly prepared mother.

In recent times there has been a surge toward breast-feeding. There are those who develop lactation or use an artificial apparatus to breast-feed an adopted child. In a study of 240 mothers who nursed their adopted children, over three-fourths of the mothers felt very positive about their experience with induced lactation.⁶ An understanding of some of the problems that women who wish to nurse their children face is presented in an article on "Closet Nursing" by Avery.⁷ These are women who breast-feed their children with some degree of secrecy, due to actual or feared intolerance from those in contact with nursing mothers.

There is error in thinking that instinct is adequate to properly prepare a mother to breast-feed an infant.³⁶ This should not be so surprising when it is recognized that chimpanzees and other animals which give birth in captivity have to be taught how to have their young suckle. Applebaum² points out that problems often develop when the physician in charge has inadequate knowledge of breast-feeding and fails to properly prepare a mother for the activity. The La Leche League International has several excellent pamphlets and a book⁵⁰ which provide proper instruction and encouragement in the art of breast-feeding. The organization also has local chapters which have educational meetings and provide camaraderie for the nursing mother.

It is necessary to understand the anatomy and physiology of lactation and the effect of milk tension letdown. When there is inadequate protractility of the nipple, there may be a lack of stimuli for the baby's instinctive response to suckle.³⁶ Procedures for correcting a flat or inverted nipple and nipple care

are presented in the La Leche material.

A comparison of the usual rubber or plastic nursing nipple and the natural nipple reveals major differences in the way a child will feed. The nipples differ in length, rate of flow, and the raised area surrounding the nipple. The softness of the artificial nipple must also be taken into consideration.⁴ In natural nursing, a baby must use his lips and incisal ridges to grasp the shorter natural nipple. The fact that there is a grasping action can be observed by pressure patterns on the incisal ridges of breast-fed infants within thirty days after birth. The activity of the orbicularis oris in this grasping action keeps the lips tight and exercises the muscles. The baby regulates the flow of milk by biting and releasing the nipple. The tongue locks the nipple against the hard palate; thus the tongue is exercised in an activity which begins the development of a normal swallowing pattern. A negative pressure develops, creating a suction for the flow of milk. The milk is then projected by the tongue from the oropharynx into the esophagus. During this process the milk is "masticated," beginning the first stage of digestion by amylase. This normal action of breast-feeding requires a backward and forward movement of the mandible during the milking act. In the infant, the digastric muscles responsible for much of this activity are approximately twice as strong as in the adult.⁶⁷ The breast-fed baby extracts approximately half the amount of milk over a longer period of time than the bottle-fed baby, and sleep comes quickly; the bottle-fed baby has to fight with over-saturation and an abundance of swallowed air. Picard goes on to state, "However, the smaller quantity of nutrition correctly received at the breast predigested by saliva is of much more value than the greater intake from the conventional bottle." The dual mechanism of suction and compression necessitates approximately sixty times more work in breast-feeding than in bottle-feeding.

The bottle-fed baby has a long nipple inserted into his mouth. It is not necessary to bite and hold the nipple with the incisor ridge and orbicularis oris. The orbicularis oris is forced open by the round base of the nipple, and it is not exercised. This begins the flaccid lip pattern of the patient with a deviant swallow. The flared, weak orbicularis oris which develops is known as Picard's mouth or bottle mouth. The flow of milk is much more rapid with less work, because the fluid is placed so far into the mouth by the long nipple.

An additional adverse factor from the rapid flow of milk inserted deep into the mouth is minimal digestive action in the oral cavity because the milk fails to mix with amylase. It is necessary for an infant



15—5. Natural nursing requires lip and biting activity to grasp and hold the nipple.



15—6. The usual artificial nipple pushes the lips apart, and there is no need to hold the nipple with lip and biting activity.



15—7. Typical mouth development as a result of bottle feeding. The weakened, flared orbicularis oris is known as Picard's mouth. Note the bilateral imbalance of the orbicularis oris, indicating different strengths of the muscles inserting into it.

Table Contrasting Natural and Nipple Feeding

	Lips	Mandible	Tongue	Chin
Natural Nursing	Sealed against gland	Closed	Elevated	Passive
Artificial Nursing	Rounded around raised portion of nipple	Open	Forward	Active

to stop the rapid flow of milk from the artificial nipple with its tongue. The flow from the usual nipple is so free that the infant could choke on the milk without some control. In some cases the mother even enlarges the opening to insure that the infant will get the milk "easily enough."

Bottle-feeding does not require the forceful action of the tongue on the palate. It is believed that this causes a narrow upper arch, with a severely contracted maxilla.⁹² There are also those who believe that finger- and thumb-sucking result from an infant's failure to adequately suckle with the increased effort needed at the breast, and from failure to receive the warmth of the nursing mother.

When it is necessary to bottle-feed an infant, a mother should be aware of the different types of artificial nipples available and choose the correct one for her child.²⁸ The three important factors of design are length, flexibility, and flow rate.

The typical artificial nipple is considerably longer than a natural one. Studies at the Institute for Myofunctional Therapy indicated that the average length of a mother's nipple is 10 mm; however, the breast is so constructed that the mother's nipple can fit into the infant's mouth and then increase in size from thirty to fifty percent during the nursing act, from 13 to 15 mm in length. Many artificial nipples, in comparison, are 1.2 to 2.1 times larger than the natural one. The mother's nipple automatically adapts to the size of the infant's mouth. The growth and consequent change in the size of the child must be taken into account when choosing an artificial nipple.

The artificial nipple must simulate the flexibility of the natural one to be able to adapt within the infant's mouth. If the size of the nipple and its flexibility cannot adapt to the infant's mouth, it becomes much like an orthodontic appliance. This is especially important at this early age when there is great potential for molding of the tissues of the oral cavity.

If the rate of flow from the artificial nipple is too great, the infant will control it with his tongue. This is thought by some to be the beginning of anterior tongue thrust. The infant should be able to control the rate of flow by the use of his facial muscles.

Those who have steadfastly held that bottle-

feeding is a primary cause of abnormal function of the orofacial muscles blame the style of nipple and large opening as the reason. Ricketts⁷³ points out another possibility which he considers speculative — an infant's sensitization to cow's milk. This can be put into perspective with our next consideration of etiology for orofacial muscle imbalance, which is breathing and airway problems. It is quite possible that an infant may be sensitized to cow's milk, creating an allergic manifestation in the upper respiratory area which causes mouth breathing and, secondarily, dysfunction of the orofacial muscles. This is certainly a possibility which should be considered.

Mouth Breathing and Airway Problems

A normal individual should breathe through the nasal cavity with the mouth closed, with little or no strain. The tongue is at rest, with no excessive force on the teeth or palate. Mouth breathing can be a result of restriction to the nasal airway, and the patient often has "adenoid facies." This is a pattern in which the head is tipped backward and there is a long, narrow face, narrow palate, open bite, and hollow cheeks. Although this pattern has been associated with enlarged adenoids, it may correlate with many other factors. There may have been nasal trauma causing chronic obstruction, allergies, congenital deformities, polyps, or neoplasms. Ricketts^{70, 71, 74} points out that "... the actual size of the adenoid is less important than the space it fills. Slight enlargement of adenoids can obstruct a small cavity, while large masses are needed to obstruct a nasal pharynx with large dimensions."

Allergy is often the cause of mouth breathing and influences the dentition.⁵⁶ In referring to an allergy, Carr¹³ states, "These children often have a congestion of the mucous membrane of the paranasal sinuses, and the openings from the nose into the sinuses are partially or totally blocked by mucous or swollen tissues most of the time. This prohibits proper aeration of the sinus, seriously hinders their development, and causes underdevelopment of the ethmoid, sphenoid, and paranasal sinuses." This underdevelopment is what causes the "adenoid facies" with its long, narrow face, narrow palate, and

open bite.

In the presence of mouth breathing, the tongue is held low in the oral cavity to increase air intake. Failure of the tongue to provide the inner muscular envelope for the maxillary area makes this individual prone to maxillary collapse and crossbite.⁷³ The mouth breather has a propensity toward tongue thrust.³⁷ Ricketts⁷⁴ observed with his speech pathologist that "... practically everyone with a stubborn tongue thrust habit exhibits breathing problems in some form."

It should be pointed out that not all mouth breathing is due to upper respiratory involvements. Leech⁵³ studied 500 patients attending an upper respiratory research clinic for diseases of the ear, nose, and throat. In this select group, only ninety-five (19%) were found to be mouth breathers. Sood and Verma⁸⁴ state that 85% of mouth breathers suffer from some degree of nasal obstruction. Twenty percent of the children between five and fifteen years of age are habitual mouth breathers.⁸⁴

In discussing tonsillectomy and adenoidectomy, Fairchild¹⁷ points out that this surgery is not beneficial to the child with nasal allergy, the thin child, and the child with occasional pharyngitis. Steele⁹⁰ states that "... if no attention is given to the child's allergic state, surgical treatment may be doomed to failure. Some patients have shown marked recession in the size of adenoid and tonsillar lymphoid tissue after several months of allergy treatment."

When an upper respiratory disturbance or allergy is a primary factor in mouth breathing, it should be taken care of before any attempts are made to change the habit. The usual approaches in applied kinesiology for allergy (described in Volume V) are frequently successful in correcting allergic or upper respiratory disturbance. Sometimes a tonsillectomy and/or adenoidectomy is necessary, but this should be done only when conservative approaches have failed. After correction, or in the absence of allergies or upper respiratory dysfunction, the standard approaches used in myofunctional therapy to balance the orofacial muscles are often successful in correcting mouth breathing. Of primary importance is obtaining normal orbicularis oris strength and length so there is an effective lip seal.

Sucking Habits

The most common sucking habit is thumb-sucking. Finger, knuckle, lip, blanket, and pacifier sucking can also be considered under this category. There seems to be no controversy as to whether thumb- and finger-sucking can cause malocclusion. Andersen¹ found 54.2% of tongue thrusters had a

thumb- or finger-sucking habit, while only 25% of non-tongue thrusting individuals had the habit. Bijlstra¹⁰ found a statistically significant correlation between finger- or thumb-sucking with sagittal overbite (protrusion). The insertion of any foreign object into the mouth displaces the tongue and keeps the teeth apart. These sucking habits do influence orofacial muscle development and occlusion; they must be corrected prior to myofunctional therapy.

Sleeping Habits

Some are of the opinion that sleeping on the back contributes to mouth breathing; they recommend a higher pillow to enhance closed mouth breathing. Huggins⁴² makes a strong case for back sleeping as an optimum approach in children. His studies developed from Stallard's concept.^{87, 88, 89}

Dewel¹⁵ finds that sleeping on one side may be responsible for lateral tongue thrust. He states that gravity may cause a sleep-relaxed tongue to flow into the interocclusal space between the posterior teeth.

Back sleeping is recommended to avoid pressures on the face and jaws which accompany side and stomach sleeping. (This concept is discussed more thoroughly in Chapter 12.)

Miscellaneous Habits

There are many other types of habits⁸⁵ and hobby and occupational activities that create problems in the orofacial musculature and with the occlusion. These activities should be recognized and brought under control, either by changing the activity or training to eliminate the habit pattern. Some of the activities involve upholsterers placing nails in their mouths and dispensing them for tacking upholstery, seamstresses cutting thread by incisal biting, and seamstresses and tailors dispensing pins and needles in a way similar to the upholsterer. Chewing toothpicks, match sticks, pipe stems, etc., throws the orofacial muscles off-balance and eventually influences the occlusion. Even worse abuse is using the teeth as a pair of pliers, such as to open bottle caps and other forms of forceful abuse. All these factors can contribute to continued problems with the orofacial muscle group, or with cranial faults.

Cranial Faults

It appears that cranial faults can contribute to deviant swallowing patterns. There have been no studies correlating this clinical observation; however, following correction improved strength has been noted in the orbicularis oris as observed on the force test described later in this chapter. There is also improvement in facial muscle strength and tongue

Myofunctional Therapy

function with the correction of some cranial faults. Apparently these muscles are disturbed by peripheral entrapment of cranial nerves V, VII, IX, and XII. It is pointed out throughout this book that the stomatognathic system is an integrated system of various complexes of muscles such as the hyoid, masticatory, deglutition, and postural groups, which must work in harmony.

Malocclusion

Another cause of tongue thrust is considered by Dawson.¹⁴ The tongue position may be a habit developed by an individual to compensate for some dysfunction. A premature contact may be so irritating — either consciously or subconsciously — that an individual develops the habit of thrusting his tongue into the area to avoid the unpleasant contact. This may also be the cause of a cheek-biting habit.

Equilibration to remove the prematurity eliminates the disturbance, if there is no other orofacial muscle imbalance; function may return to normal with no additional treatment to change the habit pattern. When a habit pattern — including that caused by dysfunction of the tongue and masticatory and facial muscles — is firmly established, re-training is often necessary to establish a correct pattern.

Palmer⁶⁴ describes the case of a dental hygienist who developed impacted wisdom teeth which were very sensitive. This resulted in inserting the tongue between the dental arches during mastication and swallowing to protect the hypersensitive area. Within three years a marked open bite developed. The effect of the tongue between the arches is to create a premature stop, throwing the force of eruption out of balance and causing tooth intrusion.

Examination

There are numerous diagnostic signs indicating imbalance of the inner and outer muscular envelopes of the teeth and a possible deviant swallowing pattern. Many of these factors can be observed during an initial consultation and further delineated during examination as body language of the condition. It is this body language which alerts the physician who is not a dentist that there may be disturbance in the stomatognathic area contributing to the symptoms, which may be remote from that initial consideration.

The discussion here is limited primarily to the stomatognathic area. This should not take away from the correlation of function in this area with the total body. Many dentists recognize the importance of integrating function in this area with the facial skeleton and muscles, closed kinematic chain of the stomatognathic system, and, finally, with the rest of the body.^{19, 30, 31, 34, 59, 66}

Some of the factors previously discussed as the etiology of orofacial muscle imbalance can be clues to the physician to further evaluate the stomatognathic area. During consultation there may be evidence of mouth breathing, history of upper respiratory disturbances, marked movement of facial muscles during swallowing, and dentalization of linguo-alveolar consonants (/s/, /z/, /d/, /t/, /l/, /n/).³⁷ In addition to these factors, more specific evaluations and observations lead to a final diagnosis to determine if myofunctional therapy is necessary. The final observation should delineate whether

tongue thrust or other orofacial imbalance is transitory, or if remedial action is necessary now. In order to make this differential diagnosis, it is imperative that the orofacial muscles be measured to determine whether a balance or imbalance of forces exists. If tongue thrust and other orofacial dysfunction are present, but without an imbalance of muscular forces, the dysfunction is possibly transitory and may correct spontaneously. On the other hand, if muscular forces are imbalanced, the condition is probably chronic, with a deep-seated engram which must be changed by therapeutic training.

Evaluation of muscular forces has been enhanced by an instrument called the Myoscanner, developed by the Institute for Myofunctional Therapy.⁴³ The Myoscanner is a force transducer which records and holds the maximum pressure applied against the transducer on a scale calibrated in pounds of force. The force transducer has been adapted to test compressive lip strength and extensor tongue strength, and to measure the mass of the masseter muscle belly. Standards have been established for the normal range for these three measurements. Deviation can be excessive or diminished, indicating muscle strength.

Measurement of muscle function is also accomplished by registering pounds of force on a force scale as a button is pulled from behind the orbicularis oris muscle. The force scale illustrated in figure 15—9 is a simple, economical instrument for clinical use which is available from Myofunctional Products.⁶⁰

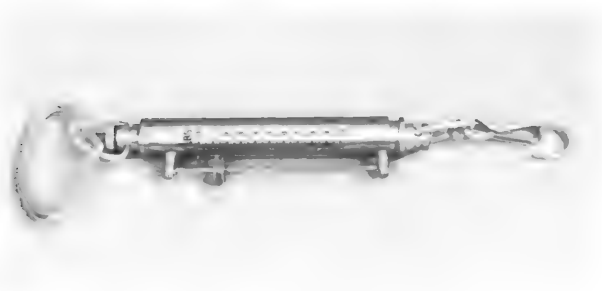


15—8. Myoscanner recorder and transducer.

Variations of instruments designed to measure orbicularis oris strength and tongue extensor force have been designed by several researchers.^{8, 48, 68}

Weak Orbicularis Oris

The open-mouth posture of a weak orbicularis oris is often the first sign of imbalanced orofacial muscles observed by the examiner. In a relaxed position the patient's lips are parted, with the upper lip flared up and the lower down. The flaring results from the muscles inserting into the orbicularis oris — primarily the levator labii superioris and the depressor labii inferioris — acting without opposition from the orbicularis oris. An individual in this situation is often a mouth breather; he may snore and have a dry mouth in the morning. The condition is more often apparent in children, because an adult has learned to maintain a closed-mouth position for cosmetic and social reasons. It will more often be apparent in adults when an individual is relaxed and alone, watching TV, driving an automobile, etc.



15—9. The force scale records the maximum amount of resistive force generated in the test.



15—10. Examiner pulls on force scale until button is pulled from behind the patient's orbicularis oris. Note the patient tilting the head forward in an attempt to compensate for weakness of the orbicularis oris.

When there is an open-mouth tendency, differential diagnosis using the Myoscanner and force scale will reveal if there is weakness of the orbicularis oris or whether a blockage of the nasal passages is causing a need to breathe through the mouth. Polyps, allergies, enlarged adenoids, or mechanical blockage are frequent causes. Standard examinations should find these.

The resistive strength of the orbicularis oris can be tested by pulling a button attached to a string from behind the patient's pursed lips. The button is standardized, approximately 25 mm in diameter. It is attached to a string which is, in turn, attached to a force scale. The examiner pulls on the force scale, which should have a built-in recorder to register the maximum amount of pull. The pull is slowly increased until the patient can no longer hold the button behind his lips. The direction of pull should be straight anterior, level with the lips. The patient should not be allowed to tilt or turn his head so that the button displaces from the central area behind the orbicularis oris. There is a tendency for a patient to shift his head when the muscle is weak, trying to recruit additional ability to hold the button. The lips should be able to resist from three to five pounds of pressure. This is not the type of test generally used in AK manual muscle testing. Because of the slow development of testing traction, the test evaluates actual muscle power rather than the muscle's ability to adapt to changing testing pressure.

Myofunctional Therapy

The Myoscanner is used to determine the compressive strength of the orbicularis oris muscle as previously illustrated. When the orbicularis oris is weak, it is usually demonstrated in both resistive and compressive strength, but not necessarily so. Sometimes one test will give evidence of muscle inability and the other test will be normal.

Generally the orbicularis oris muscles are too weak. In some cases the muscles can register too great a strength, and this may also be a problem. Throughout the muscular evaluation, the concern is normalizing the balance of the inner and outer forces developed by the muscular envelope.



15—11. Myoscanner testing compressive lip strength.



15—12. Change of compressive lip strength is monitored with the Myoscanner during myofunctional therapy.



15—13. Characteristic appearance of weak orbicularis oris.

Lip Character

Evidence of chapping — usually considered to be from lip licking — may also be evidence of lip biting, sucking, or resting the upper teeth on the lower lip. These habits are nearly always correlated with an abnormal function in the stomatognathic system.

Drawing the lips between the teeth, resting the upper teeth on the lower lip, or drawing the corners of the lips between the teeth is often done when malocclusion is present. Usually this is a subconscious body effort to avoid intercuspation when there is a sensitive prematurity or an inability of the jaws to go into proper centric occlusion as a result of muscular imbalances. This pulling of tissue between the teeth is sometimes done when there is a reduced vertical dimension. It is a method by which the body subconsciously supplies a type of bite plane to increase vertical dimension.

Facial Grimace When Swallowing

The muscles of the face should be relaxed and show no activity during swallowing.⁹² The only muscle which should reveal external activity is the masseter. Activity of the temporalis muscles is usually not observable unless specifically palpated.

When facial expression changes during swallowing, there is generally tongue thrust, orofacial muscle imbalance, and deviant swallowing. Pursing of the orbicularis oris is a natural body accommodation to counteract anterior tongue thrust. This will help maintain tooth stability because the excessive force of the tongue on the teeth will be balanced by the orbicularis oris. This is a normal body accommodation to the tongue thrust problem. The swallowing pattern should be corrected so the tip of the tongue forces into the palate, as is normal.

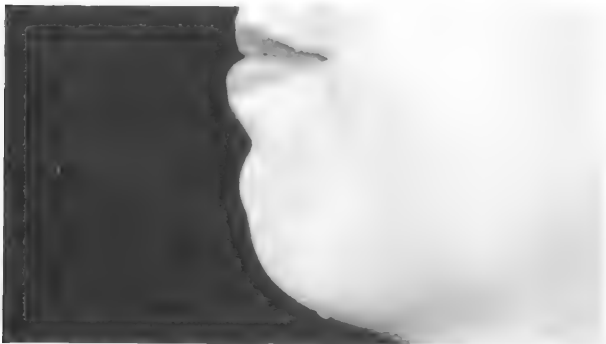
Part of the grimace may be excessive buccinator

contraction, which pulls the cheeks between the molar teeth so the mandible can be stabilized to the upper jaws without intercuspation. When this occurs, the tongue will usually have a lateral thrust between the teeth. (This is discussed further with lateral tongue thrust.)

As with other facial muscles, the mentalis muscle is normally passive during swallowing. Warrer⁹⁸ studied the amount of mentalis muscle contraction in individuals who had malocclusion and those who did not. Of thirty-one individuals who showed active mentalis function when swallowing, 90% had malocclusion. Of thirty-nine individuals who had a passive mentalis muscle during swallowing, 92% had no malocclusion. In chronic activity of the mentalis muscle during swallowing, there is a characteristic fold between the lower lip and apex of the chin when the face is at rest.



15—14. Typical facial grimace with an abnormal swallow.



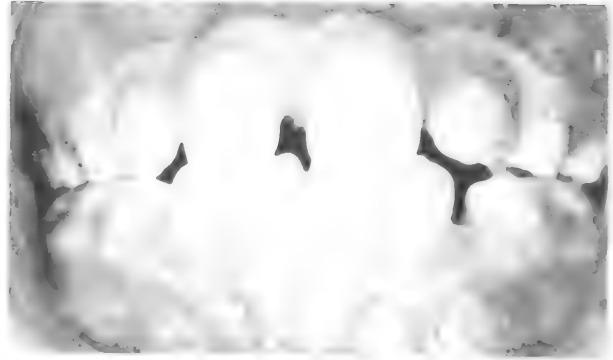
15—15. Mentalis fold, indicating hypertrophy of the mentalis muscle from chronic hyperactivity during swallowing.

Jaw Opening

As investigation progresses into the mouth, observe for mandibular deviation as the patient opens his mouth; this could be from involvement of muscles treated by myofunctional therapy.³⁵ It must be correlated with temporomandibular joint function and occlusion, discussed in Chapters 11, 12, and 16.

Diastemata

A diastema is a separation between adjoining teeth in the same dental arch. These spaces, whether in the anterior upper or lower arch or in the posterior teeth, are often caused by tongue thrust without compensating forces from the facial muscles. Further evaluation for abnormal tongue movements is indicated.



15—16. Diastemata.

Failure of Intercuspation

As has been noted, proper swallowing requires intercuspation of the teeth as the first action. Very often when there is failure of intercuspation, the tongue or cheek is placed between the teeth to provide a stable base. It is sometimes observed that the teeth are held apart without tissue being interposed between them. Rix⁷⁶ studied ninety-three children to determine the correlation between lack of intercuspation on swallowing and malocclusion. Among the children who swallowed with teeth in occlusion, 36% had dentitions which deviated from



15—17. Lateral tongue thrust which created an open bite.

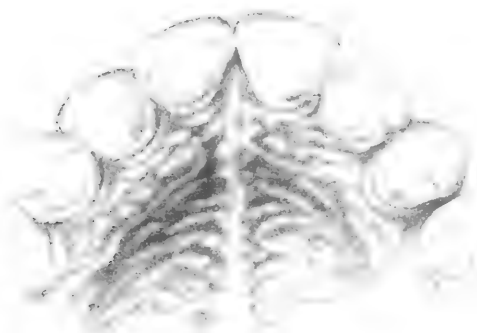
Myofunctional Therapy

normal. Of the children who swallowed without intercuspation, 81% had dentitions deviating from normal. Rix went on to state that whenever he has heard a lisp in a child, he has also found an atypical swallow.

When the tongue or cheek is pulled between the teeth during swallowing, it acts as a stop or occlusal contact for the teeth. The teeth will erupt until they meet an opposing force. When the cheek or tongue acts as this contact, the teeth fail to meet in centric occlusion. When the tongue or cheek is constantly inserted between normally erupted teeth because of an irritating prematurity or some other reason, it is possible for the teeth to intrude into the alveolar socket. This may cause loss of vertical dimension or failure of the teeth to meet in centric occlusion in that area, termed an "open bite." It will occur only if the tongue or cheek is inserted constantly with swallowing over a prolonged period.

Palate

The palate must have adequate space for the tongue. A very high, narrow palate or one distorted with torus palatinus usually indicates cranial disturbance, probably from the birth process. It can also be caused by the tongue failing to move into the palate on swallowing, thus minimizing the forces of the internal aspect of the muscular envelope. The narrow palate, whatever its cause, creates swallowing problems and indicates orthodontic consultation. Some cases of torus palatinus appear to be caused by a jamming of the intermaxillary and interpalatine sutures during skull development. It, too, may be due to the tongue's failure to press against the palate and be housed in the palatal area during swallowing. This



15—18. The convex type of torus palatinus is a ridge along the intermaxillary suture. It may result from a jamming of the intermaxillary suture during the developmental stage.



15—19. Deep rugae in the anterior palate indicate failure of the tongue to contact that area. With training, the rugae flatten out.

would cause a lack of force to spread and flatten the palatal areas of the maxillary and palatine bones.⁹² Deep rugae in the anterior palate indicate the tongue has not been contacting that area during swallowing. The palate is contacted by the tongue with each normal swallow, thus tending to flatten the palatal rugae. In the deviant swallow there is a failure of this contact, and the palatal rugae are more numerous and pronounced. Upon correction of the swallowing pattern, the rugae flatten to a more normal appearance.

Anomaly of the Tongue

A short lingual frenulum which restricts motion of the tongue is called an ankylotic tongue. It can cause disturbance in the swallowing pattern and also disturb speech, interfering with normal orofacial function. What often appears to be macroglossia is not truly an enlarged tongue; rather, it is a flat, very weak tongue that will respond to myofunctional therapy designed to activate the tongue musculature in a proper swallowing pattern.

Appearance of the Tongue and Cheeks

If there are tooth impressions on the lateral aspects of the tongue or in the cheeks, the tongue has had a lateral thrust or the cheeks have been pulled into the teeth. A ridge along the edge of the tongue also indicates placing the tongue between the occluding surfaces. The tongue which has lateral thrust will be wide and flat on protrusion, rather than the narrow, pointed tongue which develops with normal swallowing.

Tongue Position at Rest

Occasionally the tongue's abnormal position can be seen by simply separating a patient's lips and observing the tongue placed between the teeth. This

a strained, popping motion, and an elliptical hyoid motion. In coordinated swallowing the hyoid moves in a smooth circular pattern.”

Since the tongue cannot be directly observed in most cases, a procedure has been developed by Payne and Garliner,²⁵ called the Payne Technique after its basic originator. The system is to place a substance visible under longwave ultraviolet light on the tip of the tongue and have the patient swallow. The fluorescent substance transfers to the point where the tongue touches and is then viewed with the ultraviolet light shown into the oral cavity. If the swallowing pattern is normal, the transferred substance will be just posterior to the central incisors on the incisive papilla. Garliner²⁵ specifically relates how the substance will be transferred in various tongue thrust problems and types of malocclusion. It is an excellent method for initial diagnosis and following the patient's progress through myofunctional therapy.

15—20. Impressions of the teeth on the tongue are usually from lateral tongue thrust.

may be anterior or in the molar region. Since this is seen infrequently, it is necessary to have a more dynamic manner for observing tongue thrust. The Payne technique, developed by Everett Payne, D.D.S., together with the research of the Institute for Myofunctional Therapy, fills this need.²⁵

Tongue Thrust

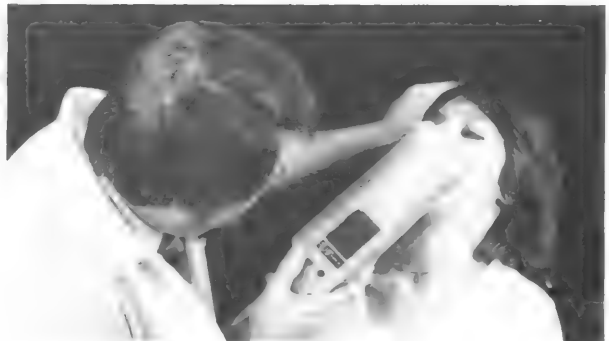
In some instances the tongue can be observed during swallowing by the examiner pulling the lips apart during the swallowing action. First it is advisable to look for areas of open bite and diastema so that observation can readily be made as to where the tongue may be thrusting. The open bite may be in an Angle Class I, II, or III malocclusion, with a lack of intercuspation because of the tongue thrusting into that area and causing tooth eruption to fail.

The patient can be evaluated swallowing saliva, liquid, or food. The examiner rests his middle finger on the cricoid cartilage of the larynx and his thumb on the portion of the lip that is to be opened during swallowing to observe for tongue thrust. The patient's swallow is indicated by a movement of the cricoid cartilage, and the examiner quickly depresses or elevates the portion of the lip to observe for tongue thrust. In some cases tongue thrust can easily be diagnosed in this manner. Occasionally the examiner will have difficulty moving the lip because it is held very tightly during swallowing. This is usually evidence of a severe tongue thrust.

Hyoid movement during swallowing can be evaluated for a smooth, circular pattern.⁸³ Ravins⁶⁹ states, “In abnormal swallowing there will be a delay,



15—21. Evaluation with the Payne Technique is done by placing a small amount of the fluorescent substance on the tip of the tongue with a toothpick.



15—22. The patient then swallows. The examiner observes under ultraviolet light where the substance was transferred during the swallow.

Habit Pattern Correction

Habit patterns such as thumb-sucking, lip-biting, and mouth breathing must be eliminated prior to or during myofunctional therapy. Ricketts et al.⁷⁵ recommend that the habit be controlled by the time the upper and lower incisors and first molars are erupting. Of course, if mouth breathing or lip-biting is secondary to other problems, the primary involvement must be corrected first. This may require correction of allergies or other restrictions to the nasal passages. As previously mentioned, lip- or cheek-biting may be due to an occlusal problem or tooth pain. In this case an individual is consciously or subconsciously bringing the tissue between the teeth to cushion the offending structure.

Thumb- or Digit-Sucking

Several studies cited by Levy⁵⁴ indicate the origin of thumb- or finger-sucking to be insufficient sucking at the breast or bottle. Goodheart³³ finds that correcting the digestive system by attention to the oral, gastric, and small intestine digestive reflexes aids in correcting sucking habits.

The approach presented here to correct thumb- or digit-sucking is one of breaking the habit pattern through the child's conscious effort. There are many types of appliances used to stop the sucking habit. Straub⁹³ recommends that such appliances not be used because they generally prevent the child from placing his tongue in the correct swallowing position. He recommends that habit therapy be instituted, and is the original developer of much of the procedure presented here.⁹⁴



15—23. Note the diminished size of the ring finger on the right hand from a chronic sucking habit.

To successfully break a habit pattern, it is mandatory that a patient desires to make the change. The therapist should explain to the patient that there will be no coercion; the procedure is one to help provide methods of eliminating the habit pattern. Most children who are thumb-suckers or have other sucking habits have been subjected to many "home remedies," nearly all of which are unsuccessful. They probably have had bad-tasting material placed on a thumb, gloves put on at night, and have been bribed and scolded, along with many other procedures. What is needed now is for them to understand that someone is available to help who has experience with this type of problem; there will be no forcing, scolding, or bribes. It is important that parents understand the procedure and refrain from scolding and bribing, giving significant support to a child during his effort. The child's guilt feelings about the sucking habit can be relieved by explaining in children's terms the difference between the conscious and subconscious mind, and relating the sucking habit to the subconscious which is causing the difficulty in control the child is experiencing.

In explaining to a child that it is necessary for him to want to eliminate the habit, it should be pointed out that if he does not have the desire the approach will be discontinued. In nearly all cases a child will readily agree that he wants to eliminate the habit. This should be verbalized by the patient without the parents present. Sometimes a child will voice a positive response when the parents are present when he really has no desire to eliminate the habit. Only occasionally is there a child who does not affirm the desire to eliminate the habit. In the case of a negative response, the therapist should state that he is sorry that the patient does not want to eliminate the habit, but without his desire there will be no therapy. In this case dismiss the child, telling him that you are sorry you cannot help; however, you will be available any time there is a change in desire. Quite often the child will call within two weeks, stating that he does want to eliminate the habit. There is generally severe peer pressure on these children and a sincere desire to establish a normal pattern; in fact, continuation of the sucking habit is often a severe emotional trauma to the child that increases with age. The most important factor in a successful outcome of the habit correction is the child's desire; the second most important factor is a perceptive, understanding, caring therapist.

The patient must be at least five to six years of age to be willing and capable of communicating with the therapist. Parents must be willing to devote the

extra time and support that the child will require for successful treatment. There are no forced restrictions or noxious substances placed on the thumb or finger to forcefully restrict the child from indulging in the habit. These procedures provide for the child who wants to break the habit a method of becoming aware of when thumb- or finger-sucking is taking place during the night. No effort is made in the beginning to correct the daytime habit; this will be taken care of later.

This first effort to correct nighttime sucking habits is the most important phase of the therapy. The parents and child are taught to wrap an elastic roller bandage over the entire length of the child's arm. Wrapping the arm does not totally inhibit the child, because elbow bending is still possible. The bandage is wrapped so that circulation is not impeded when the elbow is extended or mildly flexed. The bandage is held in place with three large safety pins. The clips that usually come with the bandage typically do not hold adequately through a night of sleep. When the elbow is flexed enough to allow the thumb or finger to reach the mouth, circulation is impeded so that numbness develops in the arm and wakes the child. Even if the elbow is flexed enough to allow the hand to approach the mouth, the afferent neurologic signaling does not meet the previous pattern of thumb-sucking, and a subconscious message will be interpreted by the child that the undesirable habit is about to take place. This is often enough to keep the child who is very desirous of abolishing the habit from sucking the digit.



15—24. Elastic roller bandage is applied in such a manner that it does not restrict the thumb or finger from reaching the mouth. It signals the child that the habit is about to occur, bringing the activity from the subconscious to the conscious mind.



15—25. Water soluble ink on the tape is smeared if the habit takes place during the night.

Two strips of adhesive tape are wrapped around the involved finger, and a few dots are made on the tape with a water-soluble felt-tip pen. If thumb- or finger-sucking occurs during the night, the dots are smeared. The child keeps a record and places a star on it for nights free of the sucking habit. Record keeping and parental approval — whether the habit correction is being conquered or not — are very important. The child who desires to break the habit will be very pleased with the accumulation of the stars. Parental support is absolutely necessary and should be given for the child's effort, not just for his success. The child also needs additional overt expression of affection and attention during this stressful period of deprivation. The mother needs to make a specific point of giving attention and affection in addition to that ordinarily given. The goal is to reach ten consecutive nights without digit-sucking. An important part of the program is a telephone call to the therapist with a report about the night. This regular interaction with the therapist and a caring parent is a mandatory part of the program.

When the child has accomplished ten successive nights without sucking a digit, the roller bandage can be removed but the tape should be retained. The tape is removed after three or four additional nights of freedom from the habit. There may be a relapse because of excessive strain at school or some other emotional trauma. If so, the last training item removed should be returned, praising the child for his continued effort in breaking the habit.

Daytime sucking should be controlled by a glove or half of a tongue depressor taped to the digit. This is used only when watching television or some other activity in which subconscious digit-sucking may occur. Again, the therapy is not that of restriction but rather of giving the child afferent stimulation that the habit is about to take place.

Myofunctional Therapy

Nail-Biting

Chewing the fingernails is not as problematic to the dentition as digit-sucking and some of the other habits. The myofunctional therapist's training and habit correction procedures are distinct advantages in providing habit therapy for these individuals. Some researchers have found that the habit is primarily psychological, and frequently it spontaneously stops at puberty.¹⁰⁰ Spontaneous remission is severely questioned in a study of 6,946 naval recruits, ranging in age from seventeen to thirty-seven. Among the men, 21.5% were found to have the nail-biting habit.⁶⁵

A method of substituting incompatible activity with nail-biting as a means of breaking the habit was studied by Nunn and Azrin.⁶² This habit reversal procedure was effective immediately in eliminating nail-biting, generally requiring only one counseling session of about two hours' duration.

Goodheart³³ finds on a clinical basis that the fingernail-biting habit is controlled more easily when trace mineral supplementation is given the patient. He also uses temporal tap as a habit correction procedure, as discussed in Volume I of this series.

Other Habit Patterns

Other habit patterns may require equilibration to eliminate a prematurity, or finding the cause of a

painful tooth. These are factors which cause an individual to subconsciously insert his tongue or cheek between the teeth to avoid contact. Some habits help balance the stresses of malocclusion which are subconsciously countered by chewing a toothpick, match stick, or some other item. Again, correcting the occlusion is necessary and may require equilibrating the teeth, balancing of muscles, correcting cranial faults, or any of the other factors which influence occlusion.

Lip-licking or lip-sucking is often a bi-product of mouth breathing. The lips become dry, creating a desire to moisten them with the tongue. The saliva, as it dries, creates chapping and leaves a sticky residue, perpetuating the problem. Lip-licking often leads to lip-sucking, which sometimes feels comfortable to chapped, cracked, sore lips. Lip-licking also is sometimes done to prevent the contact of a prematurity as described above. If present, mouth breathing must be differentially diagnosed as to cause and its correction obtained. It may be necessary to use some type of lip balm to ease lip discomfort.

Some habits deal with other physiological desires of the body, such as pipe smoking. In these cases it is necessary that the person be made aware of the problem the habit causes.

Myofunctional Therapy Exercises

There are many exercises in the armamentarium of a myofunctional therapist. Many of the procedures used today were first introduced by Walter Straub⁹⁴ who, although not the first, can be considered as the one who alerted the professions to the necessity of balancing function in the orofacial muscles.

Some of the procedures are fairly standard exercises used by most therapists; others are special exercises developed by individuals. The key factor is a therapist knowledgeable in the procedures who continually evaluates and modifies exercise procedures to meet the needs of a particular patient. Presented here are the more common exercises, to acquaint the physician with the procedures.

The description of myofunctional therapy presented here is not sufficiently in-depth to equip an individual to train patients in myofunctional therapy. The presentation is intended to alert the physician to the methods used by a myofunctional therapist for improved communication and referral ability. If one

wishes to introduce myofunctional therapy into a practice, he should attend the myofunctional workshops which are taught by the Institute for Myofunctional Therapy.⁴³

During a patient's myofunctional therapy, it is often necessary to have an observer present who will be able to aid the patient with his procedures at home, especially in the case of a child. As with any other procedure designed to change deeply-seated neurologic engrams, it is necessary to have a person coach the efforts of correction so that the procedures are done properly.

A prerequisite in beginning the therapy is the patient's desire to make the necessary changes. The primary responsibility of a therapist is to educate. This, of course, is done by teaching the exercise procedures and monitoring them, but of even greater concern is putting into perspective the importance of the task at hand. The first step toward these goals is an excellent rapport between patient and therapist,

since the two will spend a lot of time in discussion. As with any other activity, the patient is motivated to complete the procedure because of a personal desire to fulfill his needs. A child may desire to be appreciated; an adult may want to look, feel, or function better. The insight and intuitive ability of the therapist guides the discussion to most rapidly meet the patient's needs.

Before beginning myofunctional therapy, it is best to thoroughly evaluate a patient for cranial faults, spinal subluxations, and localized muscle problems in the orofacial area by applied kinesiology methods; then make any necessary corrections. When muscles get their proper lymphatic drainage, blood supply, and nerve control, they respond much more rapidly to exercise procedures. The muscles should also be evaluated for disturbance in their proprioceptors and for muscle stretch reaction to determine if there is any local dysfunction. Treatment of orofacial muscular imbalance with applied kinesiology techniques is very important in obtaining good results. Some problems with the orofacial musculature that originally appear to require myofunctional therapy will return to normal function with no treatment other than the AK approach. Most of the cases, however, are problems in improper development or long-term habits which require myofunctional therapy.

Applied kinesiology methods may be limited in results for the many types of conditions treated because of imbalance of the orofacial muscles. It may be difficult or even impossible to obtain lasting cranial corrections or improvement of some other dysfunction. Again, we are involved with the basic principle that muscles move bones, and as long as there is muscular imbalance the structural correction — no matter how effectively made — returns to abnormal because of improper muscle balance.

Mouth breathing is detrimental to effective, rapid correction with myofunctional therapy. If there is an airway restriction, it is necessary that the upper respiratory problem be corrected. Sometimes the propensity to breathe through the mouth will be from a glabella cranial fault; the patient does not have an effective option of nasal or mouth breathing (see Chapter 6). If there is no restriction to nasal breathing, it appears that an approach to enhance its use is of value. To reduce the time needed for treatment, Ravins⁶⁹ recommends a procedure for getting a patient to breathe only through his nose. He points out that the elimination of mouth breathing automatically starts an individual toward correct tongue placement for swallowing. In his procedure he recommends approaching a patient in a very friendly, warm, non-challenging way. This is done to obtain maximum relaxation in the patient, after which a mild hypnotic approach is used. Under this state he teaches the swallowing procedures. Hypnosis is used in some cases by other myofunctional therapists,⁹ but most practitioners prefer an educational, motivational program. Regardless of the method the therapist chooses, the important part is to develop a good rapport with the patient.

It is necessary in myofunctional therapy for the physician or therapist to determine precisely which muscles are weak, firing at the wrong time, hypertonic, or habitually dysfunctioning. Specific exercises for the various factors have been developed in myofunctional therapy and will be briefly reviewed here. Each individual should be evaluated for his unique needs. The Myoscanner and force scale have proven to be very effective instruments in initial diagnosis. A specific protocol should be developed for the addition of each exercise as the training progresses. Continual evaluation may show need for changing the protocol to meet the patient's needs.

LIP EXERCISES

The orbicularis oris is the keystone of the outer muscular envelope. It is often weak, reducing external force to the anterior teeth. It also influences force to the posterior arches by failing to provide a stable insertion for the buccinator; the resulting imbalance in the muscular envelope permits a tendency toward labio-version or bucco-version of the teeth. In some cases when the orbicularis oris is strengthened, there appears to be a clinical improvement in the position of the teeth. As has been stated, myofunctional therapy is not a replacement for orthodontic treatment. A study by Barber and

Bonus⁸ found that the orbicularis oris can be strengthened by exercise procedures, and that after the exercises have been discontinued, there is a lasting improvement. The increased "... muscle strength of the lips does not produce any great effect on the dentition of children exhibiting a tongue-thrusting habit and an anterior open bite with protrusive maxillary incisors." There was, however, no effort made to train the tongue to eliminate tongue thrusting.

Part of the etiology of mouth breathing is often a weak orbicularis oris, especially when there is no

Myofunctional Therapy

blockage of the airway. Strengthening of the orbicularis oris and lengthening of the lips, if necessary, help form a lip seal which encourages nasal breathing.

Often when there is prolonged orbicularis oris weakness, the muscles inserting into it become shortened. This follows the basic principle in applied kinesiology that a muscle unopposed by its antagonist contracts and may eventually shorten. Attention must be given to both the weakness of the orbicularis oris and to the shortening of the muscles inserting into it.

All the exercises presented here for lip correction will probably be needed to some degree by those who have a weak orbicularis oris. The total complex of muscles should first be evaluated for strength and length, and a specific regime established for that patient. Some of the exercises may be needed only for a brief period, while others may have to be repeated many times.

Orbicularis Oris Strengthening

Need for strengthening of the orbicularis oris is usually determined by observation of the lips and measurement using the Myoscanner and the force scale. The weak orbicularis oris allows an open position of the lips when at rest. The lips are usually pulled apart by the tight muscles inserting into the orbicularis oris. The strength of the orbicularis oris should be quantitated by testing with the force scale and button, as previously described, and with the Myoscanner. Normal resistive force tested with the force scale is three to five pounds. Compressive lip strength tested with the Myoscanner is .2 to .4 pounds for ages four to ten years, and .6 to .8 pounds for those eleven and older. These quantitated measurements are of value not only in initial diagnosis, but in following the effects of the exercise throughout treatment.

Improved strength of the orbicularis oris can often be obtained with applied kinesiology techniques. Evaluate the orbicularis oris for need of treatment to the neuromuscular spindle cell or Golgi tendon organ. Usually increased muscle strength is obtained by cranial fault correction. It appears that this improvement is obtained from improved function of cranial nerve VII. A typical improvement is for the patient to initially have one-half pound of resistive strength on the force scale, and to improve to two pounds of resistive strength immediately after cranial correction. Usually it is necessary to follow this improvement with myofunctional exercise. The important factor is that the muscle now has an improved nerve supply to respond better to the exercises.

Tug-of-War Exercise. The tug-of-war exercise is done with two buttons connected by a string, with the buttons approximately eighteen inches apart. Partners have a tug-of-war in which each one places a button behind his lips and tries to pull it away from the other. This works well with two children, or a parent can be one of the partners. For the game to be fair in a functional exercise, the pulling by both partners should be constant, without one partner quickly jerking the button from his opponent. In adult training it is often better to have the patient work against a force scale.

The pull placed on the button, whether by a tug-of-war or with a force scale, should be such that the button is pulled directly from behind the orbicularis oris. Often the patient will turn his head to lodge the button in the corner of his mouth, or tilt his head either up or down to lodge the button behind the stronger portion of the orbicularis oris. The buttons should not have a ridge so there is no irritation or cutting of the mucosa of the lips. Supplies for the tug-of-war, along with other exercises used in myofunctional therapy, can be obtained from Myofunctional Products.⁶⁰



15—26. Tug-of-war exercise.

Marshmallow Twist. Another exercise for strengthening the orbicularis oris and also giving some activity to the buccinator is to lift weights attached to the end of a string by accumulating the string in the mouth with lip-lifting activity. The type of string is important since it is accumulated in the mouth by the lips grasping it. Avoid a very thin string; use one that provides some body for the lips to grasp. The string should be twelve to eighteen inches long with a knot tied in one end, and braided rather than waxed or slick. A single plastic disk is placed on the string to



15—27. Kit for marshmallow twist exercise consists of correct string and plastic disks.

provide weight. The patient bends at the waist with one end of the string held between the teeth and the weight dangling. The lips reach out as far as possible to grasp the string and pull it toward the teeth. The teeth gain a new purchase on the string, and the weight is thus lifted repeatedly until it reaches the mouth. When this is easily accomplished another weight is added; the procedure continues until all ten weights in the kit are easily lifted to the mouth.



15—28. Marshmallow twist exercise.

Care must be taken that the patient maintains the bent-over position so the string is lifted vertically into the mouth. Some patients will make the exercise easier by extending the cervicals so the head is more upright and the lower lip can extend with the weight pressing the string against the lower lip, helping to grasp it for lifting. Garliner calls the exercise the "Marshmallow Twist" because the procedure was originally done with marshmallows as weights.

Gaining Lip Mobility

AK Procedures. As indicated above, the muscles inserting into the orbicularis oris are sometimes shortened, pulling the lips apart. These must be stretched and relaxed to gain normal lip closure. This is accomplished by exercise procedures and applied kinesiology techniques.

The muscles antagonistic to the orbicularis oris should be evaluated with applied kinesiology techniques for the need of treatment to the muscle proprioceptors, fascial release, or spray and stretch technique. The evaluation is usually done by therapy localization, then testing a previously strong indicator muscle for weakening. If muscle proprioceptive treatment is needed, it is usually the neuromuscular spindle cell; rarely is it the Golgi tendon organ.

Sometimes the muscle stretch reaction can be observed by stretching a muscle, such as pulling down on the upper lip, and then having the patient go into the test position for the muscle which was stretched, as indicated in Chapter 4. In the presence of a positive muscle stretch reaction, the patient will be unable to assume the test position as effectively as he could prior to the muscle being stretched.

The orbicularis oris and muscles inserting into it can also be tested with the muscle stretch reaction by applying the stretch and then testing with the force scale. The stretch should be done several times in different directions to test the entire orbicularis oris and the several muscles inserting into it. Most often the cause of muscle stretch reaction is a need for fascial release, probably because of the prolonged contraction of these unopposed muscles with the resulting fascia and muscle mass adhesion.

The procedures described below for muscle stretching are much more easily accomplished when the applied kinesiology procedures stated above have been performed. The exercises consist of stretching and massaging procedures. If done vigorously and persistently, the exercises alone will probably break down the adhesions of the fascia and restore the muscle proprioceptors to normal without additional help from an applied kinesiologist. It is simply observed that the therapy goes much faster with the AK procedure.

Myofunctional Therapy

Pressure Exercises. Pressure from within the oral cavity can be exerted by the patient to blow out the cheeks and lips. In order to accomplish this there must be tight contraction of the orbicularis oris, giving a two-fold value to the exercise. This forceful stretching of the cheeks and upper and lower lips can be done with air or water.

Water appears to be the best approach; it should be warm to increase circulation during the stretching procedure. First, the patient takes a mouthful of warm water and forces it into his cheeks, thus stretching the buccinator muscles. The lips are pursed by contraction of the orbicularis oris, which is naturally necessary to contain the water. This is repeated slowly five times, allowing a complete stretching action to take place in the buccinator; it is then held while the muscle relaxes. Next a mouthful



15—29. Stretch the buccal tissue by forcing water into the cheeks while maintaining tight orbicularis oris contraction.



15—30. Water forced into upper anterior vestibule to stretch upper lip.



15—31. Stretch lower lips by forcing water into lower vestibule.

of water is forced into the space between the upper anterior gingiva and the lips, pushing out the upper lip while maintaining the pursed action of the orbicularis oris. The stretched position of the upper lip, with contraction of the orbicularis oris, is held until the muscle begins to relax; this is repeated five times. The lower lip is then treated similarly by forcing water into the lower labio-gingival space five times. The lower lip area is sometimes problematic because of considerable hypertrophy of the mentalis muscle. It may be difficult for the patient to force water into this area without losing it because the orbicularis oris fails to adequately contract. Persistence with this exercise and others will accomplish the lengthening of the muscles necessary to obtain relaxed mouth closure.

The same basic procedures outlined above can be done with air forced into the various areas rather than water. Water is an improved approach because of the hydraulic effect, along with the warmth for circulation. Air has the advantage of not requiring sink facilities, so it can be done at any time. It should be emphasized to the patient that water should be used, but air is an auxiliary approach which can be practiced.

Lip Massage. Immediately after the water exercise, the lips should be stretched and massaged in the following manner. The upper lip is pulled down over the lower incisor teeth as far as possible, and the lower lip is contracted to overlap as high as possible on the upper one. The patient then contracts the lower lip against the upper and pulls it down as hard as possible against the upper lip, while lowering the mandible to draw the lower lip over the upper. This massages the upper lip, stretching it down to eventually gain relaxed lip closure.



15—32. As much force as possible should be exerted by the lower lip on the upper one as it is pulled down. This massages the upper lip, and stretches and strengthens the lower lip.



15—33. When cotton rolls are in place, the orbicularis oris must remain contracted to maintain lip pressure.

Exercising for Lip Dexterity. The muscles antagonistic to the orbicularis oris can be further stretched by distorting the mouth into every possible variation in front of a mirror. In order to pull the mouth into these contortions, there must be activity of the orbicularis oris and stretching of its antagonists. The lips should be pulled as far laterally as possible, and in all other directions which stretch the muscles antagonistic to the orbicularis oris.

Cotton Rolls. Cotton dental rolls can be placed between the gingiva and lip to provide long-term stretching. The cotton rolls are supplied in sizes 2 and 3, with the 2 more applicable for children and the 3 for adults. These are placed deep in the upper and

lower gingival-lip cavity and retained for an hour. While the cotton roll is in place, the lips should remain together with orbicularis oris contraction. This gives a double effect to the exercise. There is the stretching of the lip from the cotton roll in place, and exercising of the orbicularis oris by maintaining lip closure. Poor results are observed if the cotton rolls are inserted just prior to going to bed and are left in during the night. Orthodontists sometimes place a more permanent lip-stretcher of a similar nature to accomplish the same thing. This may be necessary for severely shortened lips that need greater persistency to obtain stretching.

TRAINING PROPER TONGUE FUNCTION

The tongue is a highly complex combination of muscles which function on an intricate basis within a movable cavity. When functioning normally, the tongue accurately repeats its actions for deglutition, speaking, and during mastication.⁴⁶ Failure of this high level of integration indicates the probable need for myofunctional therapy. Those who need the training will often lisp, bite their tongues, and, of course, have tongue thrust during swallowing.

Tongue training takes place in four stages. First, the tip of the tongue is trained to contact the incisive papilla, which is just posterior and superior to the central incisor teeth; in training this is usually called "the spot." The second stage trains the middle of the tongue to rise solidly against the palate while the tip of the tongue remains at the spot. The third step

trains the back of the tongue to dump a food bolus while the anterior portion is at the spot and the middle is tight against the palate. The fourth step is to make the swallowing pattern a habit.

Identifying the spot by sensation at the incisive papilla with the tip of the tongue is mandatory in developing a new reflex action. It is thus impossible to train an individual in correct swallowing habits if a maxillary splint is being used simultaneously, as it eliminates the afferent stimulation necessary.³²

Anterior of the Tongue

It is necessary for the patient to train the tip of the tongue to hold exactly at the spot. If held too high in the palate, it will not be in a functional position for further procedures; if held too low, it quickly relapses

Myofunctional Therapy

into the tongue thrust position. The location should be identified by the patient and the observer, both mentally and structurally. A simple drawing of a sagittal view of the palate and incisor teeth can mentally identify the spot for them. After this is accomplished, the structural identification is solidified in the patient's mind by the therapist touching the spot and then the patient touching it himself. The tip of the tongue is then moved to the spot. To a certain extent this gives sensory identification to the patient. The afferent information is enhanced by placing a five-sixteenths inch elastic on the tip of the tongue



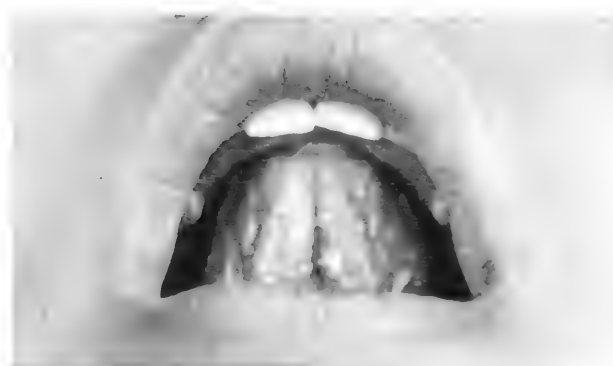
15—34. Simple line drawing made by the therapist to indicate location of the spot.

and then holding the elastic to the spot. The elastic is the type used by orthodontists and is a #6 thick-walled elastic or "crossbite" elastic. These elastics are preferred over other types because of the sharp edges which increase the afferent stimulation.

Placement of the elastic on the tip of the tongue and to the spot must be done precisely. First, the elastic is placed on the tip of the tongue as illustrated in figure 15—35. Error may be introduced by placing the elastic too far back on the blade of the tongue. The patient should be made aware that the elastic



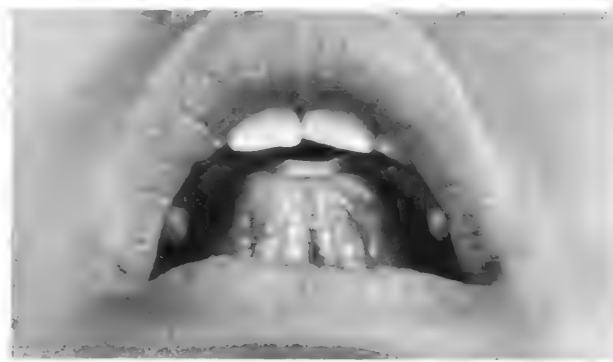
15—35. Correct placement of elastic on the tip of the tongue.



15—36. Correct placement of elastic at incisive papilla.



15—37. Elastic placed too far back.



15—38. Elastic placed too far forward.

will stay on the tip of the tongue, even though it seems very precarious in that position. If there is any tendency for the elastic to drop off, moistening it slightly with saliva will make it adhere. The patient is then asked to place the tip of the tongue and elastic at the spot, and the therapist observes its position. The position desired is for the tip of the tongue to be just slightly posterior to the incisive papilla, with the anterior edge of the elastic just posterior to the papilla. If too far back, the leading edge of the elastic will disappear; if too far forward, it will cover the incisive papilla.

With the tip of the tongue and the elastic in the correct position, the patient bites with the molar

teeth; with his lips open, he swallows. The biting begins a habit pattern of biting with the teeth together, which is the first step in a normal swallow. This intercuspation is not usually present in the tongue-thrusting individual; it must be established early in the therapeutic procedures. The lips are held open to break the reflex of the tongue coming forward to meet the lips during a deviant swallow. The lips will be held open in this manner during most of the succeeding exercise procedures. During this initial swallowing exercise, while the tip of the tongue is being held to the spot, the teeth clench and the lips open. The patient sucks in air — called a “slurp” — and swallows. This sucking component is mandatory in a normal swallow and, along with occlusal contact, must be established early in the training procedure.

A summary of the first exercise procedure is for the tip of the tongue to be placed at the spot, identified by the elastic. Teeth are closed in molar occlusion while maintaining contact at the spot. Lips are held open and the patient swallows, while simultaneously slurping. The position of the tip of the tongue should be mentally checked by the patient to determine that it has not moved; then the tongue is lowered to observe that the elastic has not moved.

When the patient has mastered this procedure, an exercise called “Mother’s Delight” is started. This is designed to train the tongue to stay at the spot. The patient holds an elastic at the spot for five minutes without talking. The rubber band produces increased afferent stimulation and an awareness of the tongue’s location. The lips must also be kept closed, forcing the patient to breathe through his nose. The procedure is called “Mother’s Delight” because it is one of the few times that a mother can keep some children quiet. The terminology can often be used as a humorous relaxation point in the therapy. Of course, for older children the title can be changed to “Boyfriend’s” or “Girlfriend’s Delight” to fit the individual.

On the next visit, the patient is thoroughly evaluated for correct procedures in placement of the elastic and the succession of events which were to take place in the first exercise procedure. Re-evaluation of each previously assigned procedure is very important in determining that the patient is doing the procedure correctly and also that he is practicing sufficiently for new engrams to be developed. Continued therapy when the patient has not mastered the previous procedure is doomed to failure. The occasional patient who cannot be motivated to follow through adequately with the procedures should be dismissed from therapy; to continue would be detrimental to all concerned. If dismissal is necessary, the therapist should express

his disappointment at not being able to continue. The patient should be made aware that therapy can be re-instituted if he desires to fulfill his obligations.

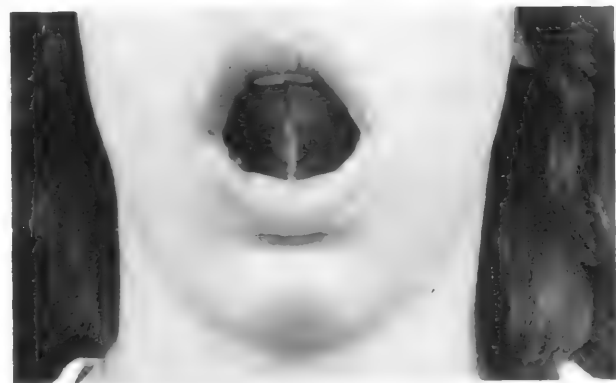
When previous procedures are adequately mastered, the next step is to do the same swallowing procedure: maintain the rubber band in position, lips open, but swallow without the slurp. This changes the activity because the slurp acts to break the reflex of the tongue moving forward to contact the lips. In other words, the slurp helps pull the tongue up and back, a natural reaction with slurping. The Mother’s Delight exercise is increased to ten minutes after one week.

Middle of the Tongue

In tongue thrust the middle of the tongue collapses rather than rises to the palate, as is proper. The purpose of this training procedure is to raise the middle of the tongue to the palate and build strength. This is accomplished with two procedures. The first is called the “hold-pull” exercise. With the mouth open, the tip of the tongue is placed on the spot; then



15—39. First step of the “hold-pull” exercise. The tip of the tongue contacts the incisive papilla and the middle of the tongue rises to the palate.

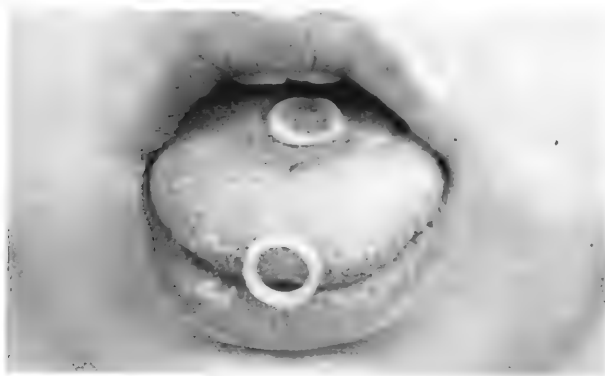


15—40. Final position of the “hold-pull” exercise. The mandible is lowered as far as possible while maintaining the tip of the tongue at the spot and the middle of the tongue raised to the palate.

Myofunctional Therapy

the middle of the tongue is raised to the palate. The mandible is then lowered while maintaining contact of the middle of the tongue with the palate. This may be quite difficult for some patients in the beginning. The mandible should only be lowered as far as the patient can still maintain the middle of the tongue to the palate. Repetition of the activity increases movement of the mandible while maintaining tongue contact.

The second exercise is a "two rubber band" swallow. One elastic is placed on the tip of the tongue as before, and the other in the middle. With the elastics in place the patient swallows. It is necessary for the middle of the tongue to rise to the roof of the mouth to hold the elastic in place. At this stage the Mother's Delight exercise is increased to fifteen minutes per day; an additional five minutes is added each week until sixty minutes has been reached.



15—41. Placement of elastics for the two rubber band exercise.

Posterior of the Tongue

Before the posterior portion of the tongue is trained, the anterior and middle must be functioning correctly, as developed in previously assigned exercises. The procedure for the posterior portion of the tongue is to learn to tip the back of the tongue to dump the bolus into the pharynx. A young patient of Garliner's gave an excellent description of a correct swallowing habit when he said, "You put your tongue up and make it act like a dump trunk." In explaining this action to young patients, the statement has come to be a common phrase in the educational process. The procedure is to have the patient masticate a small bite of protein wafer and locate the bolus in the middle of the tongue where the elastic was in the previous procedure. The patient is told to contact the tip of his tongue to the spot, bite with the



PUT YOUR TONGUE UP AND
MAKE IT ACT LIKE A
DUMP TRUCK.

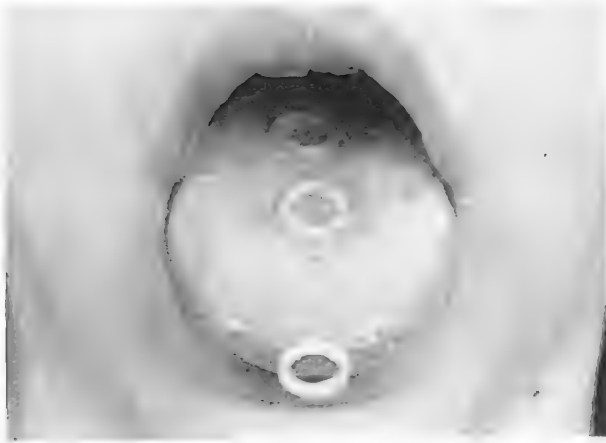
back teeth, and swallow the protein wafer with his lips open. The procedure is repeated until the patient is able to swallow food easily; he then practices at home.

The next procedure for exercising the posterior part of the tongue is initiated after the previous exercises have been mastered and progress is observed for the lips and the masseter muscles discussed below. This posterior tongue procedure gives further strength and makes it dump. It is a "three rubber band" exercise. One elastic is put on the tip of the tongue, a second in the middle, and a third elastic at the back. The patient bites with the molars, opens the lips, and swallows. This forces the middle of the tongue more significantly against the palate, and the back of the tongue tips to permit food to go into the pharynx yet stays tight against the palate, holding the rubber band.

Masseter Exercise

In most instances of a deviant swallowing pattern, there is a failure to clench the teeth during swallowing. For this reason the masseter has not been exercised and is typically weak. There will be a lack of muscle mass registered on the Myoscanner. There should be an increase of measurement as the exercise improves masseter function. Force against the transducer by the masseter muscle mass should be .4 to .6 pounds per square inch for children four to ten years of age, and .6 to .8 for those eleven and older.

The strengthening procedure is a simple isometric contraction of the masseter by clenching the teeth and holding them for a slow count of ten. This is repeated six times, three times per day.



15—43. Placement of elastics for the three rubber band exercise.

Strengthening Anterior Tongue

The next procedure is to strengthen the anterior portion of the tongue. Specific words, when said *forcefully*, will activate the front portion of the tongue. Tongue strengthening can be monitored with the Myoscanner. Protrusive strength of the tongue is .6 to .8 pounds per square inch for children four to ten years of age, and .8 to 1.2 pounds per square inch for those eleven and older. The word lists are repeated ten times a day. The first list is as follows:

too	do	loop
tip	dip	lip
tea	dear	lean
ten	did	lid
time	dime	lime



15—44. Testing protrusive tongue strength with the Myoscanner.



15—45. Measuring the mass of the masseter muscle belly.

Middle Tongue Strengthening

At this stage of the therapy the word list is changed to exercise the middle of the tongue. These are repeated *forcefully* ten times each day:

chain	jam	each
chop	jeep	peach
chew	jerry	reach
chin	jet	teach
chap	joke	much

Swallowing Training

Swallowing training initiates the program into the everyday activity of eating. The pattern necessary for proper swallowing has been taught; it must now be put into practical application for permanency.

The patient's meal is divided into halves, and one-

Myofunctional Therapy

half is eaten using the new swallowing pattern. The patient places the food in his mouth, chews it, and then the elastic is placed at the tip of the tongue and held at the spot. The teeth are then clenched, the lips opened, and the patient swallows. At a subsequent office visit the pattern is very critically evaluated by the therapist. If there is any deviation from a normal swallow, the correct pattern must be re-learned or practiced more. The food eaten in this manner is increased until the entire meal is eaten with the elastic, teeth clenched, and lips open.

Drinking Liquids

The patient is asked to take a drink of water while the therapist observes the tongue's activity. In

most cases the tongue will project between the lips. The therapist stops the patient and asks where the tongue is. In a normal swallow, the tongue automatically goes to the spot as the cup or glass is raised; in deviant swallowing, the tongue will be forward or down. The patient puts his tongue to the spot and bites with the back teeth as the cup is raised. The drink is taken by pouring the water through the teeth and forcing the tongue to do the work. With the teeth clenched, it is impossible to put the tongue between them. This therapy continues until the tongue automatically goes to the spot as a reflex action. At that time drinking can be done with the teeth apart or together.

TRAINING A PERMANENT HABIT

The procedures up to this point have taken half the intensive training period. The correct swallowing procedure has been taught, and the necessary muscles have been strengthened and patterned. It now remains to firmly establish the new procedure as a habitual lifetime pattern. There is a specific swallowing engram for each individual.⁴¹ To this point the training has disturbed the old engram, but a new one has not been developed. It will be developed only by continually using the new, yet unestablished swallowing pattern. This requires a constant reminder to the patient to use the new swallowing procedure.

Times are assigned throughout the day for the patient to check the position of his tongue and mark on a chart whether it is correct or incorrect. This causes the person to think frequently about tongue

position throughout the day. New times are periodically assigned so that the thought pattern is ultimately developed throughout the day. A chart is made by the patient to place on the dinner table to remind him of the correct swallowing pattern, since he is no longer being reminded by the elastic on the tip of the tongue.

The patient is critically evaluated by the therapist at each visit to determine if all the activities in the swallowing procedure are correct. If there is any regression, exercises are re-instituted to establish the correct procedure. Finally, a subconscious auto-suggestion pattern is given to the patient to use prior to bedtime; this helps to permanently establish a correct swallowing pattern during sleep.

Final Considerations

Again I would like to thank Professor Daniel Garliner for co-authoring this chapter. He has helped put the importance of myofunctional therapy into perspective for the practitioner whose treatments may be influenced by dysfunction in this area. I have been impressed in my study of myofunctional therapy with how important it is to have a dedicated, knowledgeable, and perceptive therapist to teach and train these procedures. The development of a lifelong habit of correct swallowing in a deviant swallower is not to be taken lightly. In Garliner's approach²⁵ the therapy lasts for one year. Intensive therapy is three months long, with a nine-month follow-up to make certain the correct pattern has been permanently established. During this time there

is continued critical evaluation by the therapist. If there is any evidence of failure to maintain the pattern, exercises are again prescribed to return the correct pattern.

This has been a brief description of myofunctional therapy to acquaint the physician with the procedures. If one wants to institute these procedures in his practice, it is recommended that the Institute for Myofunctional Therapy⁴³ be contacted for further information and training classes. Garliner's text²⁵ is highly recommended for a more thorough description of these procedures.

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Section III

Organization Between The Systems

Chapter 16

Examination of the Integrated Stomatognathic System

Throughout this text there has been an overuse of the terms “integration,” “interaction,” “influence,” “remote influence,” “relates with,” “complex,” and “interdependent.” This results from one basic fact: the stomatognathic system is an integrated complex of parts which are interdependent to the point that dysfunction of one portion can influence the total complex, which in turn interacts with the rest of the body and may cause remote malfunction anywhere within it. The stomatognathic system may also be adversely influenced by primary conditions elsewhere in the body because of its interdependence and integration with total body function. This introduction seems appropriate to Section III, which will summarize the integration of the stomatognathic system within itself and with the rest of the body. Evaluation and correction of the total complex are necessary for lasting corrections.

There has already been considerable discussion about determining the primary conditions within the stomatognathic system. In most cases, clinical examination will quickly lead the physician to those conditions in the stomatognathic system. Corrections will establish improved structural balance and physiology, and show symptomatic improvement. The more thorough search for unusual primary conditions, described in this chapter, should be used in the complicated cases where the problem is

covert. In Chapter 17 examination of the integrative function between the stomatognathic system and the rest of the body is discussed.

When the stomatognathic system is considered in its broad aspect, as Shore¹⁰ does, nearly all the structures from the shoulder girdle up are included. This is as it should be, since a change in function of any one aspect will nearly always be reflected in the function of other areas. In explaining his more inclusive interpretation of the stomatognathic system, Shore states, “An organ such as the heart or the liver can be dissected anatomically, but a system such as the stomatognathic must be studied as an integrated, physiological, functioning whole. It is important to consider the specific functions that tie this system together rather than the isolated and individual tissues which compose it.” As each individual component of the system has been described, this text has attempted to point out its integration. Before the full integration can be appreciated, a physician must first understand the system’s anatomy and physiology, and how the physical mechanism works, as well as the examination and correction for each component. With a working knowledge of the components, the time has now come to evaluate the system as a whole so that examination and correction can be most effective with minimal effort.

Examination of the Integrated Stomatognathic System

In most cases of imbalance within the stomatognathic system, only portions of the system will need correction to regain balance in the total mechanism. The key is to find primary conditions early in the investigation, since their correction will usually favorably influence many other dysfunctioning areas. If a physician enters the system and makes random corrections without any plan, much time is wasted and favorable results are delayed. The delay comes from the patient's body continually attempting to adapt to its current status. Any change taking place within portions of the system, whether from trauma or corrective efforts, initiates a chain of adaptation in other parts of the system. If a physician directs corrective effort to a portion of the system which is secondary to the primary cause, adaptation is initiated in other portions of the system which may not be appropriate for final harmony within the entire system when all aspects have been corrected. This does not mean that it is necessary to find the primary condition and treat it as the sole corrective approach; rather, it indicates that the initial examination should locate, and correction include, the primary factor. Treatment of secondary factors along with the primary one will generally speed recovery and is usually appropriate, as long as the primary problem does not remain to cause the secondary disturbances to return after correction.

The examination and correction section of this chapter includes making corrections as part of the examination process to determine if other factors cause those corrections to be lost; this helps establish primary and secondary conditions. Such progressive examination, with correction intermingled, can change a patient's status. Sometimes it is important to examine all the various components of the stomatognathic system before any treatment to document the patient's original condition for comparative purposes. It is important to specifically document objective findings in the stomatognathic system prior to treatment when a case involves an auto accident or other personal injury which could result in forensic testimony. Once corrective procedures begin, the total system starts to change in its function. If a physician is interested in the major postural muscles — sternocleidomastoid and upper trapezius — along with cervical subluxations, he may begin corrections prior to evaluating the stomatognathic area. Later in the examination it may be desirable to measure the maximum interincisal distance of mouth opening. If this is done after corrections are made to the postural muscles, cervical spine, and perhaps the cranium, there is no way of knowing if measurement of the interincisal distance at this time represents the patient's condition when he entered the clinic.

Body Language

Because of the stomatognathic system's wide range of influence on function throughout the body, many symptoms related by a patient and observations made by a physician during consultation and history-taking constitute body language indicating that the system needs evaluation. As the doctor proceeds with the examination, additional observations and findings contribute to the evidence that this system may be involved with the patient's chief complaint. Listed here is body language indicating that the stomatognathic system should be examined. The list is not all-inclusive because of the system's extremely wide range of influence over body function.

Body language is divided into cranial faults, dental conditions, neurologic disorganization, postural deviation, more general symptoms, and trauma. Individuals of various professional disciplines will be led into an evaluation of the total stomatognathic system primarily by their major area of interest.

Although the dentist is more interested in the immediate area of the teeth and related tissues, the chiropractor in posture and spinal function, and the otolaryngologist in auditory conditions, each needs to be aware of body language remote from his specialty area. Many chiropractors may hesitate to evaluate the dentition, muscles of mastication, etc., while a dentist may feel awkward having a patient stand in front of him to evaluate pelvic and spinal balance. Although we may be led into previously unfamiliar areas when evaluating the total stomatognathic system, it is necessary that we do so; otherwise, the primary condition may not be discovered, and we will be treating secondary problems and getting secondary results. As we consider the different aspects of body language, try to add other signs to the lists; no list can be all-inclusive in these interacting systems.

Examination of the Integrated Stomatognathic System

CRANIAL FAULTS

Muscle strengthening on respiratory assist	Blurred vision	Diplopia
Facial dyskinesia	Tearing (or lack of)	Masticatory muscle imbalance
Facial asymmetry	Facial synkinesis	Facial tics
Earaches	Hearing loss	Tinnitus
Poor equilibrium	Dizziness	Headaches
Respiratory symptoms	Disturbance in speaking	Dry mouth
High or low blood pressure	Digestive symptoms	Endocrine symptoms
	Circulatory symptoms	

By the time the physician concludes history-taking and consultation, he has probably obtained considerable body language information indicating cranial faults, if they are present. This presumptive evidence comes from the symptoms the patient relates, as well as from observations the physician makes while talking to him. Much of this body language is in the form of symptoms from structures, organs, or glands supplied by cranial nerves.

The first positive evidence of cranial faults is a weak muscle becoming strong as a result of respiratory assist. This may occur when the physician asks the patient to take a specific phase of respiration and a previously weak muscle strengthens. Often evidence of a respiratory assist comes from observing a patient spontaneously take or hold a specific phase of respiration in order to resist the examiner's muscle testing pressure. This body language of cranial faults should not be overlooked; it often reveals the problems whereas testing by directing the patient to hold a specific phase of respiration does not. The patient's innate effort may more effectively find the phase of respiration that puts the cranium in an optimum position.

During history-taking or consultation, specific questions directed toward a single cranial nerve or group of nerves may obtain information indicating cranial faults. Questions directed toward the eyes may gain information about cranial nerves II, III, IV, and VI, as well as about possible distortion of the bony orbit which puts pressure on the eye. Blurred vision may result when the extraocular muscles do not properly coordinate the eyes, possibly causing frank diplopia at times. Pressure on the eye from distortion of the bony orbit may cause hyperopia, which often improves significantly after cranial fault correction. Improvement may also be due to improved function of the ciliaris muscle, which modifies the shape of the lens for depth accommodation. Dryness of the eyes or excessive tearing may accompany cranial faults. The complaint of eye dryness usually comes from individuals wearing contact lenses, which are often tolerated much

better after the faults are eliminated. Improvement following cranial fault correction seems to indicate that entrapment of cranial nerve V or VII was responsible for poor lacrimal gland function.

In some instances a physician will observe imbalance of the masticatory muscles as a patient talks during consultation. This ability develops with experience in balancing the masticatory muscles and observing the before and after phonation. There are several characteristics of jaw motion during talking which are almost impossible to describe, but they become obvious to an experienced observer. Changes in phonation probably occur after improvement in both jaw motion and function of the facial muscles. Deviation from muscle imbalance can more accurately be evaluated as lateral jaw deviation when a patient opens his mouth during examination. During consultation there may be evidence of facial muscle imbalance which, if abnormal, is called dyskinesia. Facial muscle contraction can be evaluated, as described in Chapter 4, to differentiate dyskinesia from imbalanced facial expressions developed as a result of predominantly accessing right or left brain activity. Balance of bilateral brain activity is actually part of an individual's personality. It, along with other personality factors, has a major bearing on facial expression and apparent asymmetry of muscle function.

Facial synkinesis is the unintentional movement of a muscle accompanying a volitional one. This may be due to cranial faults causing peripheral nerve entrapment, but it may also result from an intermingling of axons during the regenerative process following trauma to a nerve. Facial tics may be observed around the eyes, mouth, or nose. When they respond to cranial fault correction, it appears that entrapment of cranial nerve VII was responsible.

Body language of cranial nerve VIII entrapment may be auditory, or it may relate with equilibrium and neurologic disorganization. Hearing loss and tinnitus may develop insidiously from disturbance in the stomatognathic system; the problems may be acute as a result of trauma or rapid change in the dentition.

Examination of the Integrated Stomatognathic System

There may be associated earaches and frequent ear infections. Dizziness or poor equilibrium indicates possible involvement with the vestibular division of cranial nerve VIII.

Some patients with imbalance in the stomatognathic system develop speech irregularities, such as a change in tone, a lisp, or difficulty in controlling volume. Generally a patient will comment about this change, which followed an accident or other etiology. Because of the complex activities of speech, several cranial nerves may be involved. Accompanying

speech difficulty may be a dry mouth or excessive salivation.

Many symptomatic patterns develop with vagus nerve dysfunction. Whenever there are complaints of digestive or respiratory symptoms, or perhaps circulatory disturbances, cranial faults should be considered.

Symptoms relating with the endocrine system may result from improper nerve supply or the cranium's relationship with the pituitary, pineal gland, or hypothalamus.

DENTAL CONDITIONS

Malocclusion
Denture problems
Bracing
TMJ pain

Tooth loss
Periodontitis
Bruxism
Jaw popping or crepitation

History of dental procedures
Periodontosis
Severely worn teeth
Discomfort when chewing

It may be difficult for a non-dental physician to observe some of the intricacies of malocclusion. Quite often this is not necessary if the doctor listens to the patient and asks the proper questions. If the subject is brought up, the patient will often remark, "My teeth don't fit right since the accident." Often malocclusion is very evident, even to the non-dental physician. Evaluation should include observation for tooth loss or severely worn teeth which may be responsible for loss of vertical dimension.

During history-taking there should always be a question about previous dental procedures. There may be a close time correlation between the beginning of chronic headaches and the performance of major dental procedures, such as prosthetic crowns, bridges, etc. Full mouth extraction with subsequent dentures is a shock to the stomatognathic system and may result in the beginning of imbalance in the system. On the other hand, a patient may state that his health has improved since the extractions. Improvement may be attributed to the loss of "rotten teeth," which it may well be; however, it is possible that the dentures improved balance within the stomatognathic system, and by a chain effect improved cranial function, hyoid neuromuscular balance, etc. Further evaluation of the stomatognathic system should be done.

Periodontitis may relate with poor oral hygiene or cranial faults. The necessity of good oral hygiene for the prevention of periodontitis is well established. Most authorities agree that the trauma of malocclusion can cause periodontitis or create an environment in which it can develop. A less recognized prodromic factor of the condition is poor tissue health secondary to peripheral entrapment of cranial nerve V. Periodon-

tosis is a degenerative, non-inflammatory condition in which there is destruction of the periodontal tissues. It may result from malocclusion or poor tissue health, as well as from many other disturbances in the stomatognathic system. Periodontitis may eventually develop in these tissues.

Bracing and bruxism may be psychogenic or secondary to disturbance in the stomatognathic system. Evidence of these conditions is jaw tenderness, severely worn teeth, hypertrophied muscles, complaints from a spouse of grinding the teeth at night, or clenching on awakening in the morning.

When there is discomfort from chewing, it is obvious that the stomatognathic system should be evaluated. Another less recognized sign of stomatognathic dysfunction is when a patient's symptoms develop after eating. Usually the physician attempts to correlate this with the type of food eaten and with digestive function. Sometimes the symptoms develop because of stress to the stomatognathic system from chewing. If there is malocclusion, chewing may cause temporary cranial faults or otherwise aggravate the status of the stomatognathic system. Several hours later when the stress subsides the symptoms improve, only to recur during the next masticatory process.

Popping or crepitation in the temporomandibular joint is always evidence of a disturbance in the stomatognathic system. Unfortunately, many physicians discount this evidence if it is asymptomatic. This is true of many other body language signs relating to the stomatognathic system. We all tend to discount that which we do not thoroughly understand.

NEUROLOGIC DISORGANIZATION

Positive TL to K27
General evidence of dis-
organization

Positive TL to CV24 or GV27
Right and left brain imbalance

Ocular lock

Neurologic disorganization as discussed here is known in applied kinesiology as switching. Switching is discussed in Volume I, and amplified and correlated with the stomatognathic system in Chapter 18 of this text. In this discussion it will suffice to state that whenever switching is indicated, the stomatognathic system should be evaluated. Applied kinesiology indications are positive therapy localization to K27, CV24, or GV27. The relation of cranial faults with ocular lock — an inability of the eyes to function properly together — should be obvious.

General evidence of neurologic disorganization is seen when an individual does exactly opposite that which is intended. This is exemplified when an individual lies down on his stomach when he is requested to lie on his back, turns right when asked to turn left, etc. Neurologic disorganization is also evidenced by a significant imbalance between right and left brain function, shown when a strong indicator muscle weakens when a patient says the multiplication tables or hums a tune.

POSTURAL DEVIATION

Head not level
Recurrent cervical fixations

Spinal curvature
Cervical kyphosis

Recurrent cervical subluxations
Pelvic imbalance (category I,
II, or III)

Because the stomatognathic system is so important in neurologic organization throughout the body, it should be evaluated whenever there is a postural deviation, such as an unlevel head, shoulders, or pelvis. Spinal curvature has been correlated in applied kinesiology with neurologic disorganization, which is nearly always present in these individuals. Cervical spine disturbance should especially be

related to the stomatognathic system because of the system's postural muscles which support the spine at this area. This can manifest as recurrent cervical subluxations or fixations. When a cervical kyphosis is observed on x-ray, it is nearly always secondary to imbalance of the stomatognathic system's postural muscles.

GENERAL SYMPTOMS

Sinusitis
Hypertension

Headaches
Shoulder/arm syndrome

Facial pain

Several types of general symptoms and conditions should automatically alert a physician to evaluate the stomatognathic system. Sinusitis may be directly related with cranial faults, or it may be secondary to an allergic condition which also may be partly caused by cranial faults. Headaches and facial pain have a wide range of etiology resulting from stomatognathic system dysfunction. Hypertension, too, is often secondary to cranial faults, but it should be recognized that there are numerous other causes

of it and of headaches.

It is usual to think of cervical subluxations as a possible cause of a shoulder/arm syndrome. Dysfunction in the stomatognathic system can cause the postural muscles of the cervical spine to be responsible for vertebral dysfunction in this area. The muscular imbalance can also be responsible for other aspects of imbalance in the thoracic outlet creating a shoulder/arm syndrome.

TRAUMA

Blow to head

Cervical "whiplash" dynamics

Trauma — a blow to the head, or cervical "whiplash" dynamics — is a positive indication that the stomatognathic system should be evaluated. These direct injuries can be recent, or almost-

forgotten episodes. Trauma throughout the body can secondarily influence the stomatognathic system; this is discussed in Chapter 17.

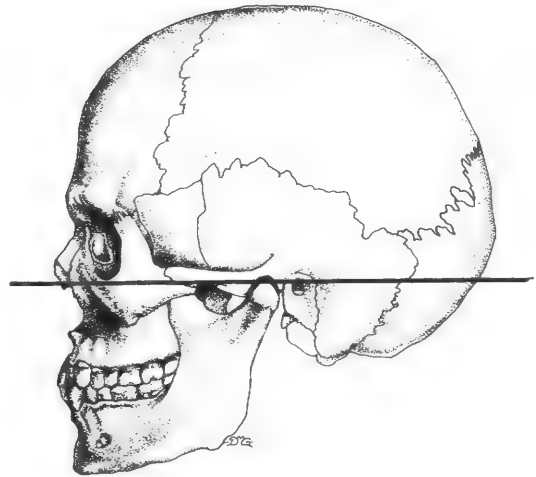
Sequential Steps of Examination and Correction

When evaluating the stomatognathic system, the importance of a level head cannot be overemphasized.⁴ One of the primary reasons this is so important is probably because of the activity of the jaw muscles from stimulation of the proprioceptors in the upper cervical vertebrae. Funakoshi and Amano³ demonstrated that tilting of the head in decerebrated and labyrinthectomized rats caused increased electrical activity in the ipsilateral masseter, temporalis, and digastric muscles. The responses in the muscles were abolished after the first three cervical nerves were cut. It seems from this that the body-on-neck reflexes may have a direct influence on the muscles of mastication in the presence of an upper cervical subluxation, fixation, or postural distortion. A level head refers to lateral tilt as well as to anterior-to-posterior balance. When the head is not level, it is often part of a total spinal and pelvic distortion. In this chapter we assume that the shoulder girdle is level and functioning normally, giving the stomatognathic system a solid base from which to function. If the shoulder girdle, pelvis, and other areas of the body are imbalanced, the additional information presented in the next chapter should be correlated with the stomatognathic system.

Observation for a level head is relatively easy when considering lateral head tilt. Structures which are ordinarily symmetrical on the two sides of the body, such as the eyes and ears, are compared to see if they are level with each other; the midline should be vertical. Most individuals are asymmetrical,^{1, 8, 14} which should be taken into consideration when evaluating structural balance. Occasionally there is enough skull asymmetry that the eyes and ears are not level when the head itself is; consequently, all structures should be taken into consideration before a final determination is made. The position of the head should also be compared with the cervical spine. If the spine is straight as viewed from the anterior or posterior, and the head is not level, there is indication of an occipital or upper cervical subluxation as part of the imbalanced complex. Usually the cervical spine and head will deviate as a curved or straight unit.

It is more difficult to determine if the head is balanced anterior to posterior. The orbitomeatal plane was designated as a standard, normal horizontal plane of the head by an anthropological congress in Frankfurt, Germany, in 1884. The "Frankfurt plane" is marked on x-ray through the upper margin of the external acoustic meatus (porion) and the lower margin of the left orbit (orbitale). These points can be closely located on a subject being examined

without x-ray by palpating the lowest point of the infraorbital rim or the orbitale. The posterior point is the midpoint on the upper border of the entrance into the external auditory meatus. This closely matches the porion.^{2, 7}



16—1. Frankfurt plane from the porion to the lower margin of the left orbit.



16—2. Frankfurt plane.

The sternocleidomastoid and upper trapezius are the primary postural muscles of the stomatognathic system. The deeper muscles of the upper spinal column, some of which insert into the skull, also give considerable support within the system's closed kinematic chain. Imbalance of any of these muscles may be a primary cause of disturbance in the stomatognathic system, or their imbalance may be secondary to dysfunction in another part of the system.

When the shoulder girdle is level but the head is not, there is usually imbalance of the sternocleidomastoid, upper trapezius, or deep spinal muscles. Very often the primary cause of these muscular imbalances is vertebral or occipital subluxations or fixations, or cranial faults influencing cranial nerve XI. Although remote body dysfunction, such as a foot subluxation, can cause a head-leveling problem, shoulder girdle imbalance and other postural deviations will usually be coincident.

When the shoulder girdle is level but the head does not parallel it, the postural muscles of the stomatognathic system should be evaluated for weakness, hypertonicity, or shortness. Very often the imbalance will be found in the sternocleidomastoid and upper trapezius muscles. These muscles are intimately associated with the stomatognathic system and are nearly always involved to some degree when there is imbalance within the system.

A better knowledge of the sternocleidomastoid and upper trapezius is gained from understanding their unique nerve supply. They are the only postural muscles of the body receiving both cranial and spinal nerve supplies. The sternocleidomastoid receives its efferent supply from cranial nerve XI and its major afferent supply from the anterior rami of the 2nd and 3rd cervical nerves. The trapezius also receives its efferent supply from cranial nerve XI; its afferent supply is from the ventral primary divisions of the 3rd and 4th cervical nerves. This makes these muscles uniquely vulnerable to dysfunction as a result of cranial faults, upper cervical subluxations or fixations, and gait problems.⁶ Cranial nerve XI may be disturbed by either subluxations, fixations, or cranial faults because the cells of origin are in the posterolateral part of the anterior horn of the spinal cord, from which the nerve travels through the foramen magnum to exit from the cranium at the jugular foramen. There are apparently some afferent fibers to the muscles from cranial nerve XI,¹⁸ with their cell bodies in the intercranial portion of the nerve trunk.^{9, 17}

Interference with the sternocleidomastoid or upper trapezius nerve supply almost insures a major disturbance in the balance of the closed kinematic chain of the stomatognathic system, and confusion within the nervous system. If a cranial fault changes the efferent supply, resulting in a hypo- or hypertonic muscle, the afferent supply will reflect the muscle imbalance; however, it will probably not be acted upon appropriately because of the efferent supply's inability to properly control the muscle. Adaptation begins within the closed kinematic chain to attempt to modify the disturbance. For illustrative purposes, consider a subluxation of the occiput with the atlas creating peripheral entrapment of cranial nerve XI as

it traverses the foramen magnum. The result may be hypertonicity of the upper trapezius. There will be one of two results if this occurs: (1) the head will not be level, resulting in neurologic disorganization, or (2) the body will set adaptation in motion, the magnitude of which can only be speculative.

If the head is unlevel from imbalance of the upper trapezius, as in the example above, confusion of the body's orientation in space will result. The labyrinthine and visual righting reflexes report the unlevel condition of the head, which probably cannot be reacted upon because of the peripheral entrapment of cranial nerve XI. The body's inability to correct the head imbalance requires adaptation to take place in the stomatognathic system's closed kinematic muscle chain. An attempt to balance the structure may require increased action of the hyoid and masticatory muscles to balance the abnormal hypertonicity of the trapezius. Since the trapezius is hyperactive on only one side, increased action of the hyoid and masticatory muscles will probably be on only one side. This can cause malocclusion, in turn possibly creating additional cranial faults, or at least eliminating the natural effect of a good occlusion improving cranial function. The changes in function which occur are probably wide and varied among individuals. Clinical experience does not reveal a pattern; rather, it is characterized by diverse reactions which occur in individuals who appear to have the same primary cause of a problem.

In this illustration where an occipital subluxation is the initiating factor, only entrapment of cranial nerve XI has been considered. It is possible for the receptors of the body-on-head reflex, located in the ligaments of the upper cervical vertebrae, to be improperly stimulated as a result of the subluxation. This stimulation to the afferent system increases the confusion within the nervous system, requiring additional attempts by the body to return to normal function. The great diversity of receptors which can be stimulated from this type of imbalance is the reason there is no standard pattern for which one can look to determine a method of correction. The ability of applied kinesiology evaluation to locate improper stimulation to the afferent system and determine how it is affecting the body provides an optimum method of determining the correction needed. Cloacal synchronization and PRYT techniques, as well as correction of neurologic disorganization, are prime examples of the applied kinesiology approach to correcting these disturbances (Chapters 7, 17, and 18).

Prior to correcting cranial faults or spinal subluxations or fixations relating with postural muscle imbalance, it is best to examine the muscles for any

Examination of the Integrated Stomatognathic System

local disturbance and correct it, if present. This follows a basic principle in applied kinesiology that structural deviation is often caused by muscular imbalance (function determines form). The subluxation, fixation, or cranial fault may be secondary to muscular imbalance, or at the least maintained by it. This is applicable whether the muscular dysfunction is primary and created the subluxation or cranial fault, or is secondary to one of them. It seems that subluxations and muscular imbalance perpetuate each other. A cranial fault, subluxation, or fixation may cause a nerve to inappropriately control a muscle, which in turn abnormally supports the bony structure and maintains that distortion. In addition to correcting the bony structure, treatment should be directed to the muscle(s). This may require any of

the techniques used in applied kinesiology. It may be necessary to stimulate the neurovascular, neurolymphatic, or some other reflex. The meridian system may require balancing, or a chemical factor — such as nutrition — may be required. Often the treatment needed for a local disturbance in the muscle is fascial release or spray and stretch technique, as well as correction of proprioceptor dysfunction, which aids in obtaining correction of the skeletal disturbance. When trauma to the muscle is the etiology of the imbalance, local treatment to the muscle will often correct the skeletal disturbance and no further treatment is needed. In any event, it is consistently evident on a clinical basis that examination and local treatment to the muscles enhance corrective efforts to the skeletal aberration.

MUSCLE PROPRIOCEPTORS

The neuromuscular spindle cell and Golgi tendon organ in the postural muscles of the stomatognathic system are frequently disturbed by trauma. This is often seen in the hyperflexion-hyperextension cervical injury (whiplash) where a muscle is severely stretched. The traumatized proprioceptor can usually be palpated as a fibrous mass in the belly of the muscle (neuromuscular spindle cell), or at the musculotendinous junction (Golgi tendon organ). It will

usually be very tender and demonstrate positive therapy localization. The muscles are treated in the usual manner (Volume I) with two-point digital pressure spread apart over the neuromuscular spindle cell, or digital pressure over the Golgi tendon organ toward the belly of the muscle to strengthen a weak muscle. The digital pressure is reversed to reduce hypertonicity.

MUSCLE STRETCH REACTION^{5, 15}

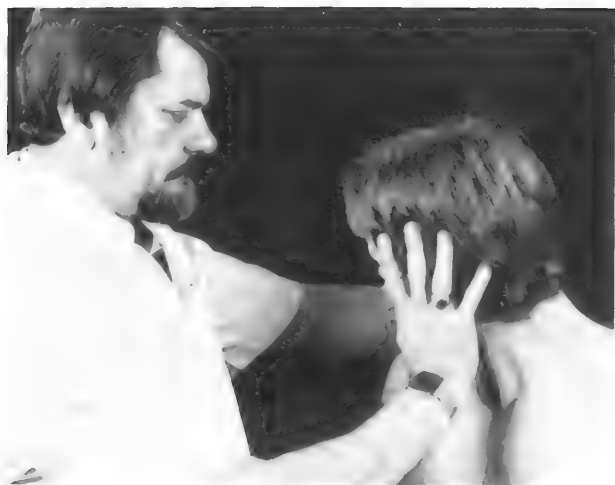
The postural muscles of the stomatognathic system usually exhibit the muscle stretch reaction as a response secondary to a weak antagonist muscle, or a chronic postural deviation where a muscle is shortened. The test is performed in the usual manner; the previously strong muscle is stretched and then re-tested for weakening. As usual, care should be taken not to excessively stretch the muscle since that normally causes temporary inhibition.

There is another reason not to vigorously stretch the muscle. If it inserts into the cranium, it can give a false positive test by challenging a cranial fault. There should also be differential diagnosis of a muscle stretch reaction from possible cervical subluxations or fixations, which may be challenged with cervical spine movement when stretching the muscle.

The sternocleidomastoid muscle is best stretched

with the patient seated. First the muscle is tested, taking care that the patient is adequately stabilized and in the correct starting position. During the test, the patient should not be allowed to tilt or turn his head. These movements recruit the deep cervical flexors and may result in an erroneous finding. The muscle is best stretched with the head turned approximately 30° away from the side to be stretched. The examiner then moves the head and neck into extension and contralateral flexion to the side being tested. The exact stretching procedure will vary slightly between subjects. The examiner visualizes the muscle fibers and directs his stretch toward maximum lengthening of the muscle. The sternocleidomastoid muscles are dominant in fast fibers; therefore, a somewhat rapid stretch is more likely to obtain a positive response.

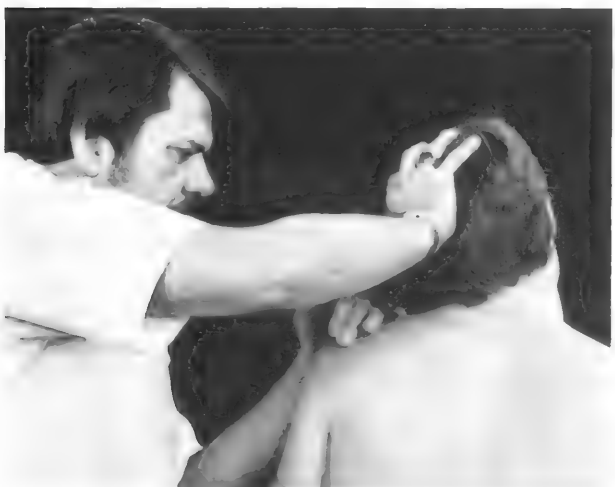
The upper trapezius is also best tested for



16—3. Test of sternocleidomastoid muscle. Note the examiner's stabilization of the patient.



16—4. Stretch the sternocleidomastoid, and then immediately test the muscle for the muscle stretch reaction.



16—5. Starting position to test the upper trapezius is with the patient's head turned slightly away from the side being tested.



16—6. The patient's head is turned toward the side being stretched.

muscle stretch reaction with the patient seated. The starting position for the test is with the head rotated slightly away from the side being tested, and the neck and head laterally flexed toward the elevated shoulder. If the muscle tests strong, it is then stretched by having the patient rotate his head slightly toward the side being stretched. The examiner depresses the shoulder and moves the head and neck into forward and lateral flexion. The head should not be turned away from the side being stretched, as it is in the muscle test, because the chin approximates the shoulder girdle and blocks the stretching ability in most individuals. Immediately after the stretch, the

muscle is re-tested for weakening. The upper trapezius, being a postural muscle, is more dominant in red fibers and responds more often with a muscle stretch reaction to a slow stretch.

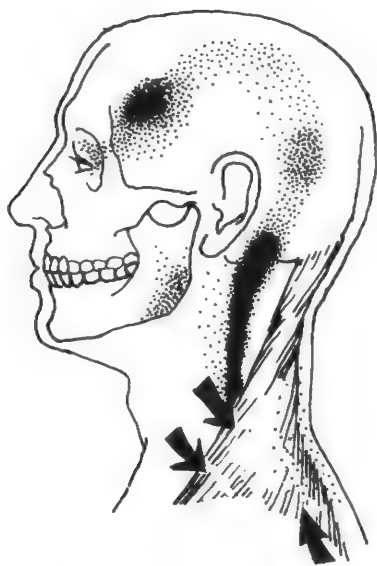
When there is a positive muscle stretch reaction, the applied kinesiology treatment is spray and stretch for trigger points, or fascial release technique. Trigger point treatment is usually indicated when there is associated pain; fascial release technique is more often needed when there is greater shortening of the muscle. It is possible that both types of treatment may be necessary.

Examination of the Integrated Stomatognathic System

Trigger Point Evaluation

Trigger points in the upper trapezius and sternocleidomastoid often refer pain into more central areas of the stomatognathic system, which may confuse diagnosis. Pain may appear to be localized in the jaw, teeth, or some other area; in reality, it is caused from the trigger point. It seems that the trigger points develop secondary to hypertonicity in the muscles, which in turn can be secondary to an imbalance in some other area of the stomatognathic system. It has been observed that trigger points may spontaneously disappear when a primary problem is corrected. It is best to locate the trigger points and direct treatment to them for the most rapid return to normal function.

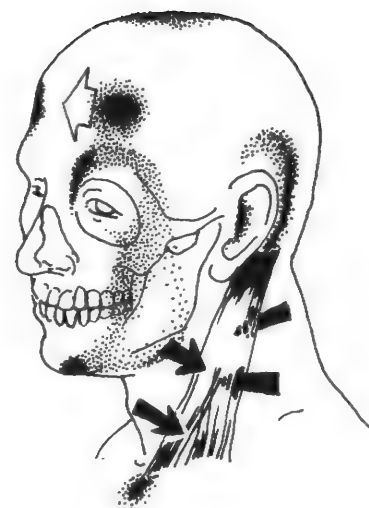
Travell¹² describes trigger points in the supra-scapular portion of the upper trapezius muscle. These points refer pain to the posterolateral aspect of the neck and mastoid process, the temple, and the back of the orbit, and some pain to the lower molar teeth and the occiput. Another area where trigger



16—7. Trigger points in upper trapezius and supra-scapular areas radiate pain to the stippled and solid areas.

points are commonly found is in the musculature of the upper cervical area. These may be in the upper trapezius or in the deep cervical musculature. Pain is referred to the eye and forehead, and sometimes to the maxillary and occipital areas. These locations are not mapped by Travell, but they are commonly seen in a clinical practice.

Trigger points can be located in almost any area of the sternocleidomastoid muscle.¹² They refer pain to the forehead, supraorbital ridge, and inner angle of



16—8. Pain radiation from trigger points in the sternocleidomastoid muscle. Illustrations 16—7 and 16—8 from Travell,¹² *Journal of Prosthetic Dentistry*, with permission.

the eye, middle ear and posterior auricular region, point of the chin, and pharynx; some pain may radiate to the cheek and occasionally to the molar teeth. Pain referred to the forehead may cross to the contralateral frontal region. Trigger points in the inferior aspect of the sternal division can refer pain to the sternal area. Travell^{11, 12} also relates trigger points “ . . . from the sternal division (to cause) blurred vision, homolateral lid lag, lacrimation, and coryza; from the clavicular division, homolateral sweating of the forehead, postural dizziness, and imbalance.” In light of current understanding of the neurologic disorganization which can result from stomatognathic system imbalance, it is difficult to know where the dizziness related by Weeks and Travell¹⁶ to trigger areas in the sternocleidomastoid muscle actually originates.

Fascial Release Technique

When a muscle stretch reaction is positive and no trigger points are found, or if the reaction is still present after trigger point therapy, fascial release technique should be applied. It is most often needed in the upper trapezius and deep posterior spinal muscles; occasionally it must be applied to the sternocleidomastoid and deep cervical flexors. After

application of the technique, there should be an increased range of motion, and the muscle stretch reaction should be abolished.

Muscles which exhibit the stretch reaction are usually secondary to weak antagonist muscles. When treatment of the upper trapezius muscle is required, the contralateral upper trapezius and the ipsilateral lower trapezius should be evaluated, as well as the neck flexors. In case of the sternocleidomastoid or deep neck flexors exhibiting a stretch reaction, the cervical extensors should be evaluated for weakness.

If the head is not level and examination does not reveal weakness, hypertonicity, or shortness of the postural muscles of the stomatognathic system, further evaluation is indicated. There will usually be a remote problem in the body for which the imbalanced head is compensating. It may be in the gait mechanism, cloacal synchronization, PRYT patterns, pelvic misalignment, foot dysfunction, or any of other numerous correlating structures or functions. This indicates that the patient should be examined for conditions discussed in Chapter 17 on integration of the stomatognathic system with the rest of the body.

CRANIAL CORRECTION

The next step after leveling the head is to evaluate for and correct cranial faults. This may have already been necessary in order to balance the sternocleidomastoid and upper trapezius muscles in the process of leveling the head if there was disturbance in cranial nerve XI. Correcting cranial faults early is an optimum approach because of the tremendous change it makes in the stomatognathic system. Eliminating entrapment of cranial nerve V will probably improve function of the masticatory muscles, which may change the occlusion or the way the masticatory muscles pull on the cranium. The neuromuscular balance of the hyoid is under the direction of cranial nerve XII. It, along with cranial nerve VII, is responsible for the orofacial complex. All of these muscles are part of the stomatognathic system's closed kinematic chain. Attempting to balance them when the cranium is not functioning normally becomes a frustrating and futile experience.

Cranial faults will nearly always be present when there is general imbalance in the stomatognathic system. A specific effort to define them and obtain correction at this stage will save much time as the examination progresses. Therapy localization may miss the presence of a cranial fault. It appears that for therapy localization to reveal a fault, the patient's fingers must be placed exactly over a suture that is sufficiently strained to abnormally stimulate the nerve receptors within the suture. Certainly, general therapy localization will occasionally reveal a cranial fault, but not consistently. A better approach is to challenge the structure; this will reveal cranial faults when therapy localization does not. It will also save time, because when therapy localization does find an apparent cranial fault it is still necessary to challenge to confirm that the therapy localization is actually

indicating a cranial fault. Challenge is also necessary to determine precisely how the cranial fault should be corrected.

One area of therapy localization that does appear to be effective, and determines if cranial faults are present when they might not be readily found otherwise, is over the pterygoid plates of the sphenoid and the pyramidal process of the palatine bone. This area consistently appears to be in strain when major cranial faults are present.

As stated in Section I, the optimum approach to enter the cranial mechanism for correction is with inspiration and/or expiration assist faults when they are present. Very accurate vectoring of the challenge to find the maximum indicator muscle weakening provides information for application of the optimum corrective force. When these cranial faults are accurately corrected, the cranium's closed kinematic chain is activated to effectively correct many additional cranial faults on a secondary basis.

The next most effective cranial correction is for internal or external frontal faults. Pressures applied for correction of these faults put force into the closed kinematic chain of the cranial mechanism through the facial bones directed to the sphenoid, a key bone in cranial motion.

In a high percentage of cases it will be found that no cranial faults remain after accurate correction of the inspiration and/or expiration assist cranial faults. When faults do remain, another high percentage of total correction will be obtained with internal or external frontal fault correction. Patients who have faults remaining after these corrections are those with more resistant, rigidly locked skulls which require persistence in obtaining optimum cranial function.

STOMATOGNATHIC AREA

The next area to examine and correct for expedient balancing of the stomatognathic system is the stomatognathic area, which includes the temporomandibular joint, muscles of mastication, and the occlusion. At this stage correction will be directed toward the temporomandibular joint and muscles of mastication, if they are dysfunctioning. This area is important as the next step because of its potentially adverse influence on the cranial primary respiratory mechanism. This discussion assumes the reader has studied Chapter 14.

It is best to first do screening tests on the muscles of mastication and correct them, if necessary, because tests for occlusion, swallowing, etc., may cause loss of cranial correction; if the jaw closer muscles are corrected, they may not.

Therapy localize the temporomandibular joint without occlusion to determine that it tests negatively. If the test is positive, further investigation of the joint itself should be done to determine the cause of positive therapy localization. If the TMJ therapy localizes negatively, have the patient open and close

his mouth in the sagittal plane while therapy localizing the TMJ and test a previously strong indicator muscle. If positive, continue the testing procedure to determine the involved muscle(s) and make correction. Next, have the patient stretch his jaw forcibly open while therapy localizing the TMJ, then test a previously strong indicator muscle. If positive, proceed with further testing to determine which muscle is hypertonic or shortened and make the appropriate corrections. Make certain that the stretching test did not cause any cranial faults to return.

With the above examination and corrections completed, the stomatognathic area can be further evaluated for malocclusion. Immediately after the forced centric occlusion test, re-evaluate the cranium for recurrent cranial faults. If there were muscles elsewhere in the body which strengthened after correcting cranial faults, re-testing these is a quick way to determine if the cranial faults have returned. If so, correct them again and proceed with the hyoid evaluation, cautioning the patient not to bite down.

HYOID

Sometimes correcting the neuromuscular balance of hyoid suspension will eliminate what appears to be malocclusion or muscular imbalance causing the return of cranial faults from the centric occlusion test previously mentioned. This appears to be due to a reactive muscle disturbance of the masticatory muscles from one or more of the hyoid muscles.

A simple screening test — challenging the hyoid both right and left — will find many hyoid muscle involvements. If the screening test is positive, it usually localizes the muscles involved to the right or the left side. If a previously strong indicator muscle weakens when the hyoid is moved to the right, the muscles involved are on the left side, and vice versa.

When hyoid neuromuscular balance is normal or

has been corrected, re-evaluate the cranium to determine that faults have not returned during hyoid examination and correction. Repeat the forced occlusion test to determine if it still causes a recurrence of cranial faults; if it does, further evaluation of the stomatognathic area is in order, and it should be done with the thoroughness described in Chapter 14. This includes mandibular movement in the transverse plane, various movements for muscle hypertonicity or shortness, and sequential movements for reactive muscles. If there is still a problem with recurrent cranial faults, or other disturbance within the stomatognathic system after the forced occlusion test, the integration of the total stomatognathic system should be examined.

Integration of the Stomatognathic System

The examination and correction procedures described to this point will correct most cases of imbalance in the stomatognathic system. This does not mean that these procedures may not need to be repeated. Depending upon the scope of the imbal-

ance, they may need to be repeated numerous times for adaptation to take place and new engrams to be built. In our laboratory, we have observed on the Mandibular Kinesiograph that the typical new patient, prior to treatment, has reproducible mandibular

motion and rest position regardless of prematurities, abnormal jaw motion, or increased-decreased rest position. After instituting corrections to the stomatognathic system, the reproducibility of mandibular motion and rest position is initially no longer present. After adequate time for adaptation, a new engram is built and reproducibility returns.

At this point there is no consistent method by which to determine the length of time and number of treatments necessary for adequate adaptation to occur. As a general rule, more time is required when there is a rigid skull, malocclusion, severe postural deviations, and TMJ disc involvement. The more severe and numerous these problems, the greater the length of time to regain normal function. Another factor, discussed in the next chapter, which adds considerably to the required corrective effort and time for adaptation is numerous remote problems in the body which adversely influence the stomatognathic system.

The usual procedure for treatment is to schedule a patient for examination twice a week or weekly. Too frequent scheduling does not allow adequate time for adaptation to take place. This is evident when it is necessary to work with a patient more frequently because he has traveled hundreds or thousands of miles for treatment, and available time before he returns home is limited. These patients usually have many problems throughout the body. It is best to not work with the stomatognathic system every day, but to progressively clear the various conditions which present themselves, allowing enough time for adaptation to take place after the corrections. This is achieved by rotating examination and treatment so that the same system is not treated every day. For example, the stomatognathic system may be the primary objective during one office visit, and a foot and gait problem primary on the next visit with no examination of the stomatognathic system. This still presents the problem of not allowing enough

time for adaptation because of the influence the foot and gait have on the stomatognathic system.

When a patient returns for additional examination and treatment of the stomatognathic system, the same evaluation outlined above should be repeated. Good evidence that the body is accepting the corrections, making adaptations, and building new engrams occurs when an examination reveals different indications for correction. For example, on the initial visit the indicator muscle weakened with therapy localization to the temporomandibular joint on jaw opening. Further evaluation found that the external pterygoid required muscle spindle cell treatment. On the next evaluation a week later, there is still indication of a jaw-opening problem; however, the external pterygoid is normal, and the indication is to treat the anterior belly of the digastric on the right. On the next evaluation there may be no indication of the jaw-opening problem; however, the hyoid neuromuscular balance may need treatment. On subsequent visits fewer positive findings appear; finally the entire system tests normally. This shifting of positive findings appears to be due to stresses in the stomatognathic system changing as the condition is corrected. It ultimately develops homeostasis with the adaptation of the engram and structures to normal function. During this adaptive process the patient's original symptoms are usually relieved. In some cases different symptoms develop but are short-lived. These appear to be due to the changing stress throughout this complex system as adaptation toward normal occurs.

If subsequent examination reveals the same abnormal finding that appeared to be corrected on the previous visit, some other undiscovered factor primary to the condition is present. It is at this stage that the more thorough evaluation of the cranium, temporomandibular joint, hyoid mechanism, and postural factors should be considered.

Troubleshooting the Stomatognathic System

If corrections are not obtained and maintained with the procedures outlined thus far in this chapter, each component of the stomatognathic system should be evaluated with the thoroughness described in its respective chapter. In addition, the influence of factors remote in the body, as described in the next chapter, should be considered. If there is still recidivism, the stomatognathic system should be evaluated for reactivity among its components.

There is a reason for every persistent dysfunction; the question is whether the physician is capable of finding it. Quite often when a condition persists, the answer to the problem is outside the physician's area of expertise. This is why it is important for him to be knowledgeable in other specialty areas, at least for a screening examination, so that appropriate referrals can be made when necessary.

It is relatively simple to test for reactivity of one

Examination of the Integrated Stomatognathic System

complex from activity in another. It is time-consuming, but fortunately it is not often required.

The area with a recurring problem is considered the reactive or target area. The examination is to determine the component and type of dysfunction causing the reactivity. To illustrate the principles used in this type of examination, let's consider a case where the patient's symptoms are digestive in nature, which the physician considers to be due to cranial faults. This is apparently an accurate assumption since the patient obtains relief from the digestive disturbance after treatment, which may last for one to several days. Now the stomatognathic system must be more thoroughly evaluated to determine why the corrections are lost.

Each time the patient returns there is head tilt to the right, secondary to left upper trapezius weakness and cranial faults. Correction of cranial faults and fascial release on the right sternocleidomastoid level the head. Evaluation has shown that chewing does not cause recurrent cranial faults. There does not appear to be any hyoid imbalance, and for the sake of this evaluation we will say there is no problem below the shoulder girdle.

The major recurrent problem is the weak trapezius or the recurrent cranial faults; either could be primary. Weakness of the upper trapezius followed by tightening of the contralateral sternocleidomastoid, causing both muscles to pull in an imbalanced manner on the cranium, could create cranial faults. On the other hand, the cranial faults could be primary, causing peripheral entrapment of cranial nerve XI and resulting in both the weakness of the upper trapezius and the hypertonicity of the contralateral sternocleidomastoid. Each individual component of the stomatognathic system has been thoroughly evaluated and found negative. The only recurrent problems continually observed on each office visit are the head tilt and cranial faults.

Before the examination to determine if the activity of one component in the stomatognathic system is reactive to another, it is necessary to first examine the total mechanism and correct any deviations from normal. In this case it is necessary to again correct the cranial faults, which strengthens the upper trapezius, and do a fascial flush on the shortened contralateral sternocleidomastoid.

Sometimes body language will provide clues about the component causing the problem. A way to find these clues is to ask the patient what type of activities are done before symptoms develop. In our example no abnormal body language is evident, and various components are tested randomly to determine if they cause either cranial faults or weakness of the upper trapezius. In this case it is logical to test the

upper trapezius for weakening because it is always strengthened by correcting cranial faults; thus the test evaluates both the muscle and the return of cranial faults. First the patient is given various substances to chew which are usually non-allergenic, such as almonds, carrots, or cheese; no weakening of the upper trapezius is evident. Various facial muscles are then contracted, such as in a strong smile, frown, grimace, etc.; there is no weakening of the upper trapezius. Next the major postural muscles of the stomatognathic system are contracted against standard testing pressure, and the upper trapezius is immediately tested. This is standard applied kinesiology reactive muscle testing as is done elsewhere in the body. No weakening is observed. No hyoid neuromuscular imbalance has previously been found using general indicator muscles. Now the indicator muscle used is the upper trapezius. When the hyoid is challenged by moving it to the left in a superior and posterior direction, the left upper trapezius weakens. Since the hyoid was previously challenged in this direction and found negative when the tensor fasciae lata was used as the indicator muscle, the challenge is repeated and that muscle is tested and found negative. This brings up the point that sometimes a general indicator muscle will not reveal a problem. This hyoid challenge is selectively causing a reactivity of the upper trapezius. The muscle probably involved in this direction of challenge because of being stretched is the right omohyoid. Its evaluation with therapy localization indicates a probable neuromuscular spindle cell dysfunction. It could be corrected at this time, but that would not give the physician positive evidence that it is responsible for the recurrent cranial fault. To further evaluate the omohyoid as a possible cause of the recidivism, the patient is asked to forcefully contract his muscles for right and anterior neck flexion. The patient then returns to the neutral position and is tested for left upper trapezius weakness; it is present, as are the cranial faults. The patient is allowed to rest for a short time, and then is re-evaluated for the trapezius weakness and cranial faults; they are still present. It can be hypothesized that the reason for the recidivism is upper trapezius weakness, which is only present immediately after major contraction of the omohyoid muscle. In this head and neck motion of lateral and forward neck flexion, the omohyoid and the sternocleidomastoid contract, and the contralateral upper trapezius weakens. The pull on the cranium from the sternocleidomastoid is not resisted by the upper trapezius, and the cranial faults are re-established. The cranial faults appear to cause entrapment of cranial nerve XI, which results in continued weakness of the upper trapezius and

solidly maintains the cranial faults. It can be further hypothesized that after treatment, which corrects the cranial faults and upper trapezius weakness, several days may pass before the patient makes a forceful move that contracts the omohyoid in exactly the manner which re-establishes the cranial faults. This is why he obtains one or two days' relief from his digestive disturbance after each office visit.

Another example illustrates a different reason for recidivism. The patient is a school-age child with a primary complaint of headaches. The headaches are chronic, with some relief obtained by medication. The headaches have been blamed on eye strain, and a visual examination reveals myopia and slight esophoria. Prescription glasses have not relieved the headaches, but the child's vision has improved. Applied kinesiology examination reveals numerous cranial faults, hyoid muscle imbalance, and a significant head tilt. The patient is evaluated without her glasses with an ophthalmic telebinocular and is found to have 70% vision for nearpoint and 88% for farpoint. Leveling the head and correcting the cranial faults and neuromuscular balance of the hyoid improve the vision to 90% nearpoint and 96% farpoint immediately after treatment. On a subsequent visit the parent informs the physician that the child had no headache for the rest of the day after the treatment, and awakened the next morning free from pain. Upon returning home from school, the child again complained of a headache. In five subsequent visits similar reports are given to the physician — relief from the headache, but it returns. At this stage the patient is returning to the office with no hyoid muscle imbalance and her head level. Each time there are cranial faults, although their nature seems to be changing. The specific pattern — the headache's return after a day in school — prompts the physician to question the child about school activity. Thorough questioning about physical activities during gym reveals no potential problem. The child is not active in any sports or other activities after school. When questioned about posture during classroom work, she says that she does not slump in her seat, but sits upright when listening to the teacher or writing. She does admit to propping her head on her hand when reading. At this stage of the office visit all corrections have been made and the child is ready for dismissal from the day's treatment. Before leaving she is given a book and a desk and told to read as she does in school. She puts one elbow on the desk and rests her head on her hand, basically contacting the zygomatic and maxillary bones. The physician tests for cranial faults and there are none. The child is then asked to read for approximately five minutes. When the physician returns and tests for cranial faults, they are

present as they were during the initial office visit. Habit correction eliminates the recurrent problem.



16—9. *Leaning habits can cause recurrent cranial faults.*

This points out the necessity of evaluating a patient's habits and occupational activities which might re-create the problem. When a patient obtains temporary relief from therapy but the condition returns, the right approach is evidently being followed but something happens to re-create the problem. Habitual and occupational stresses are most easily found by questioning a patient about his activities just prior to the return of symptoms.

One additional example will help solidify the investigatory activity a physician must exercise in order to find the cause of recidivism.

Sometimes a series of events must occur before a patient loses a correction. Suppose the stomatognathic system is diagnosed as the primary cause of a patient's intense neck and shoulder pain. The postural muscles of the neck are severely imbalanced, with resulting subluxations in the cervical spine and distortion in the shoulder girdle which causes neurovascular compression at the thoracic outlet. Correction of the cranium and cervical spine results in balancing the shoulder girdle and producing satisfactory relief from pain. The patient then goes several days without any pain; however, it always seems to recur after heavy lifting, a necessary part of his job as a heavy equipment mechanic. The patient recognizes this as the cause and explains to the physician that the shoulder and neck pain develop within a few hours after a heavy lift. In order to locate

Examination of the Integrated Stomatognathic System

the source of recidivism the physician has already gone through the steps of reactivity testing as previously discussed, but he is unable to re-create the cranial faults and cervical subluxations. With normal function in the stomatognathic system, he has the patient attempt to lift the hi-lo table. Being a rather strong individual, the patient is able to lift the heavy end of the hi-lo; he states that it is a lift comparable to that which re-creates his shoulder problem. When the stomatognathic system is tested, no disturbance is found. The patient is dismissed, with no insight into why the condition recurs after a heavy lift.

The next day the patient phones and reports that within two hours of leaving the office, his neck and shoulder pain returned. He is asked to return for further evaluation. The test the previous day — lifting the hi-lo table — indicates that the lifting by itself is not responsible for the return of the cranial faults and cervical subluxations. The physician next queries the patient about what he did when he went home. The response is, "Nothing. I ate supper and watched television. While I was watching TV the pain returned." This does not seem to be a contributory factor, but the physician recognizes the need to test the patient during the same activities that re-created the problem. Again the stomatognathic system is examined and all corrections are made. The patient is asked to lift the hi-lo table once more and is tested for return of any disturbance in the stomatognathic system; none is observed. The patient is then given some almonds to chew to simulate eating supper, and the test is repeated. This time cranial faults and imbalance of the stomatognathic system's postural muscles are found. This re-creates part of the problem, but no cervical subluxations are found, so the patient is asked to sit for a while and read, simulating watching television. When the physician returns from taking care of another patient, the cervical subluxations are present. Since much effort has been expended in trying to correct this patient's problem, and the Workmen's Compensation case is dragging beyond ordinary limits, the physician decides to repeat the series of events and observe more closely, attempting to discover the mechanism causing recurrence of the cranial faults, muscle imbalance, and subluxations. Again, all corrections are made in the stomatognathic system, and the patient is asked to lift the hi-lo. This time very close observation reveals a strong grimace on the patient's face during the lifting procedure. No other unusual act is observed. Again there is no return of cranial faults, muscle imbalance, or subluxations after the lift; however, immediately after chewing the almonds the cranial faults and muscle imbalance return,

followed a few minutes later by the subluxations. Some sequence of events must be occurring to cause the condition to return.

It is necessary at this stage to begin dissecting the activities to find exactly what is taking place. Again all corrections are made to the stomatognathic system, and individual factors are evaluated. First the patient is asked to chew the almonds; no disturbance is noted. The lift itself does not seem to be the cause of the problem, since no disturbance in the stomatognathic system is found after it is performed. Although the patient has previously been tested to see if facial muscle contraction causes recurrence of cranial faults with negative results, the physician repeats the test, this time having the patient add a very strong facial grimace to simulate that observed during the lift. No recurrence of the problem in the stomatognathic system is observed. The patient is then asked to chew the almonds, and immediately the cranial faults and muscle imbalance develop. The physician hypothesizes that the very strong facial grimace disturbs the cranium in such a manner that the upper dental arch is distorted; however, the cranial distortion is not recognized as a cranial fault in the manner in which the patient has been tested, nor does it impinge on cranial nerve XI to cause the muscle weakness.

To test this hypothesis, the physician has the patient therapy localize to the intermaxillary suture; the test is positive. To further test the hypothesis, he corrects all previously known factors in the stomatognathic system; the intermaxillary suture no longer shows positive therapy localization. With a strong facial grimace, the positive therapy localization returns. Now the physician has the patient simply bite down hard, and the cranial faults return. This supports the physician's hypothesis. The facial muscles composing the grimace are tested by therapy localization and palpation. Several of the muscles are treated with neuromuscular spindle cell and fascial release techniques. The patient is again tested after the strong facial grimace; no positive therapy localization develops at the intermaxillary suture, nor do cranial faults return after biting down hard or chewing almonds. The patient is then asked to lift the hi-lo table, using the same facial grimace, and is tested through the series of events which previously caused the cranial faults, muscle imbalance, and cervical subluxations to return; the test is negative. The series of events is explained to the patient, and he is advised to develop a habit pattern of using no facial grimace with heavy lifting. There is no further recurrence of the neck and shoulder problem.

This example is not hypothetical but represents a

case report. It strongly points out the need to evaluate patients in the manner in which they live. Too often patients are evaluated for the physician's convenience. Most problems do not develop when a person is lying on an examination table, and most stress to the body is not present in that position; yet that is the position in which a patient is most often examined. In problem cases it is necessary to re-create the daily activity corresponding with the development of the problem. There is a reason for everything that happens. The question is, do we have a large enough data base of knowledge to systematically search out the primary cause? In the last example, the facial grimace probably developed as a natural body effort to attempt to improve long-standing cranial dysfunction. Note that individuals who have cranial faults will take a deep breath or hold a certain phase of respiration just prior to lifting. This is regularly seen during manual muscle testing as a patient catches a phase of respiration to resist the examiner's pressure. The use of a facial grimace for this purpose becomes an habitual act. Although the patient's cranium had been corrected, the facial

grimace remained; constant overuse of the muscles was probably responsible for apparent neuromuscular spindle cell dysfunction and shortening of the muscles.

As has been stated before, most disturbances in the stomatognathic system are corrected with the quickly performed examination and correction procedures described earlier in this chapter. It is only when the condition persistently returns that it is necessary to dissect the system's activities and integrate them with the patient's daily activities to find the answer to the problem. Usually when the answer to these difficult cases is found, the treatment is something very simple, such as in the last example. It is necessary to work with the system, not against it. One of the goals of optimum cranial therapeutics is that the treatment not be iatrogenic. These examples aptly illustrate how simple the cause of recidivism can be when the primary problem is actually found. It is not necessary, and may actually be harmful, to try to force the skull into improved function. When conditions return, it is necessary to work smarter, not harder.

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Chapter 17

Stomatognathic System Organization To Total Body

There has been some discussion of the importance of correlating the stomatognathic system with total body function, but most of the consideration until now has been of structure from the shoulder girdle up. With an understanding of the stomatognathic system's intricate function, it is necessary now to relate it with potential dysfunction throughout the body.

The previous chapter stressed how dysfunction in one part of the complex can be primary to dysfunction in another area. The same basic principle is present when dysfunction of remote body parts influences the stomatognathic system, and also when the stomatognathic system adversely influences remote areas in the body. It has already been discussed how function throughout the body can be disturbed as a result of entrapment of cranial nerves; that will not be repeated here. The major endeavor

will be to understand the interactions of the body with the stomatognathic system, and how to examine for and correct resulting dysfunction.

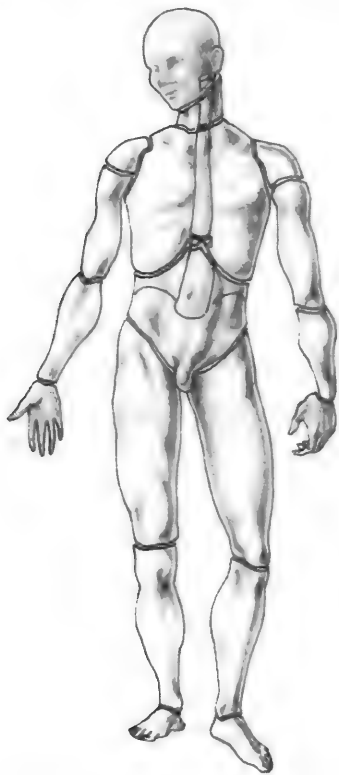
In Chapter 16 it was assumed that the shoulder girdle was level. With that base from which to work, the first consideration was to level the head. Very often the shoulder girdle is not level; thus there is not a stable base from which to work in balancing the stomatognathic system's structure. Leveling the various body modules is primary, in addition to leveling the head. Much of this is discussed in Volume I, where the basic procedures of applied kinesiology are considered. This chapter will emphasize how some of the basic procedures influence the stomatognathic system. The subjects of gait, weight bearing, and the body's modular activity will be expanded, with particular reference to their influence on the stomatognathic system.

Modular Organization

Structural balance of the body can best be considered in modules. A module may be composed of a complex such as the neck and head of the stomatognathic system, or the pelvis, thorax, and shoulder girdle of the trunk. Further, each section of a complex can be considered as a module within the complex. During the first observation of structure,

imbalance of the major modules is most striking. When the major module off-balance is the neck and head complex, further dividing it into its own modules (such as the neck and the head) can help determine the structure responsible for the imbalance. For example, if the deviation is primarily between the head and neck, there is indication of an

Stomatognathic System Organization To Total Body



17—1. The major body modules illustrated here can be further subdivided.

upper cervical disturbance; if it is of the entire neck and head module, postural imbalance from the major muscles — sternocleidomastoid and upper trapezius — is indicated. The same is true of the various divisions composing the major trunk module.

Evaluation of modular organization begins with postural balance, discussed primarily in Volume I. A patient is evaluated with a plumb line anterior to posterior, posterior to anterior, and laterally. Obvious distortions may be observed in this postural analysis, at which time the major postural muscles related with the distortion are tested; appropriate applied kinesiology treatment is done if they are weak or hypertonic.²³ A typical example of this type of structural imbalance which may influence the stomatognathic system is a unilaterally weak latissimus dorsi

which allows shoulder elevation on one side. This shoulder girdle imbalance creates a poor base from which the stomatognathic system must function. Therapy localization and/or challenge may find any of many factors as the cause of the weak latissimus dorsi. There may be an active neurolymphatic reflex, neurovascular reflex, vertebral subluxation, meridian system imbalance, or even disturbance in the cranial-sacral primary respiratory system.

Unlevel shoulders may be the result of adaptation to a more inferiorly imbalanced structure. A weak gluteus medius muscle allows the pelvis to elevate on that side, in turn causing the shoulder girdle to also elevate on that side. It follows that the head will often be tilted, with elevation on the side of the weak gluteus medius.

In addition to evaluating the major modules of the body in the coronal and sagittal planes, the arm, leg, scapula, foot, and hand positions are observed. Muscles which may be responsible for imbalance of these structures are evaluated and appropriate corrections made. The patient is then examined for leg length and pelvic function as in a category I, II, or III. It is seldom that a true short leg is present in the absence of fractures or trauma to the epiphyseal plates during growth. As the body modules are balanced and normal neuromuscular activity returns, what appeared to be a leg length imbalance will nearly always be corrected.

Correction of major muscular imbalances, pelvic and thoracic subluxations, spinal subluxations and fixations, and the cranial-sacral primary respiratory system will correct many modular imbalances within the body. Further applied kinesiology testing procedures determine if organization between body modules and activities is normal. Among these are PRY technique, gait testing, and cloacal synchronization (discussed in Volume I). Cloacal synchronization technique is further amplified in Chapter 7 of this text regarding its correlation with the stomatognathic system. PRY technique is amplified here as PRYT technique, and gait testing is related to the stomatognathic system.

PRYT TECHNIQUE

PRY technique, an acronym for pitch, roll, and yaw, is discussed in Volume I. Goodheart has added another factor — tilt — to this modular organization of body function, changing the acronym to PRYT.¹³ There has also been further description of the yaw pattern, giving it three divisions.¹² This discussion assumes that the reader knows the PRY technique

as described in Volume I. A brief outline of the examination and treatment techniques is presented, with a discussion of the newer phases as they are added to the outline.

PRYT technique relates to the interaction of body modules. Positive findings appear to result from improper stimulation of proprioceptors, primarily of

the equilibrium division. Corrections are administered to areas known to be high in proprioceptors. The ligaments of the upper cervical area relate with the body-on-head reflexes.¹⁶ Treatment to correct pitch and tilt requires hard contraction of the cervical muscles, which is an area that has a higher concentration of neuromuscular spindle cells than anywhere else in the body.¹⁹

The examinations used require the positioning or activating of two areas simultaneously. Positive findings are indicated only when an indicator muscle weakens with both modules activated at the same time. For example, examination of pitch requires flexion of the pelvis and the head and neck at the same time. A positive finding occurs when a previously strong muscle weakens. When the muscle weakens with only pelvic flexion or neck and head flexion, it is not a pitch problem; rather, it is a localized involvement in the pelvis or neck which should be corrected prior to evaluating for pitch. Factors which might be confused with a PRYT problem are mentioned, along with the various examination procedures used in PRYT technique.

Pitch

A disturbance in pitch is seen as poor organization of the major body modules — the head, shoulder girdle, and pelvis — on a flexion or extension basis. From a postural consideration, this is viewed from the lateral aspect.

Flexion. Pitch-flexion is examined with the patient supine. Any previously strong muscle can be used as an indicator. Most clinicians test the pectoralis major (clavicular division) bilaterally during these procedures. If these muscles are used, care must be taken that the patient does not flex at the elbow, recruiting the biceps brachii into the test. First the patient is asked to lift his head and touch his chin to his chest. While the patient is in this position, the indicator muscle is tested for weakening. If it does weaken, there is some type of problem in the neck, which may be a subluxation, a fixation, or possibly a local muscle disturbance; the problem should be corrected before further evaluation of modular interaction. If there is no weakening, the patient is told, "Let your head down and put your feet on the table." With the hips and knees thus flexed, the indicator muscle is tested. If it weakens, there is a problem of some type in the pelvis, hips, or perhaps lumbar spine. The local area should be examined and correction obtained.

The testing procedures so far have ruled out, or caused to be corrected, individual problems in the cervical spine or pelvis relating with flexion. Now the pitch test can be accomplished, which is simply

repeating the actions in the previous two tests simultaneously. With the patient's chin toward his chest and his hips and knees flexed with his feet on the table, an indicator muscle is tested. If there is weakening when these are tested together but none when they are tested individually, there is evidence of a pitch problem. This appears to be due to incompatible simultaneous motion of the two modules.

Apparent dysfunction is consistently found in the motion of the cervical spine. Correction is the same as that used for occiput-on-atlas flexion type of rocker motion. The physician places his hands on each side of the head, rigidly holding it in position as the patient strongly attempts to flex his head on his neck by pulling his chin toward his chest, but not lifting his head off the table. This is repeated four or five times.

Extension. A similar examination procedure is done to evaluate for pitch-extension. First the patient's head and neck are placed into extension and a strong indicator muscle is tested to determine that there is no local problem. If there is, it is corrected before continuing. The head is returned to a neutral position and the hips are placed into extension by dropping the legs over the edges of the table if it is narrow, or by placing a pillow under the pelvis and lower back. Again, the indicator muscle is tested to determine if there is a local problem. When both areas are negative during individual tests, the two are combined and the indicator muscle is tested for weakness. Disturbance in function appears to always be in the cervical spine-head relationship.

Correction is the same as that used for occiput-on-atlas extension. The physician uses broad contact on both sides of the head to resist any motion, and the patient attempts to strongly extend his head on his neck with as much force as possible. This is repeated four or five times. After correction of either pitch-flexion or extension, the patient should be re-evaluated to determine the effectiveness of the therapeutic attempt.

Flexion and Cervical Rotation. A third type of pitch coordination problem is tested in the same way as pitch-flexion, but an aspect of yaw — explained later — is added. In the presence of a negative pitch-flexion test while the patient has his neck, head, knees, and hips flexed, he is asked to turn his head to the right and the previously strong indicator muscle is again tested. If it weakens, there is a positive pitch-flexion and cervical rotation pattern if there is no weakening on head turn only. The test is done both right and left. To correct, the physician holds the head on both sides and has the patient attempt to strongly turn his head while the motion is resisted by the physician. This is done four or five times in the

Stomatognathic System Organization To Total Body

direction of the positive test. After correction of any of the three types of pitch pattern involvement, the patient should be re-evaluated to determine the effectiveness of the therapeutic attempt.

Roll

Roll refers to modules of the body not being level when viewed from the anterior or posterior. It appears to relate with organization between the proprioceptors of the visual righting reflex and those of the pelvis, particularly in the sacroiliac articulations. Treatment to the sacrum simulates the Logan Basic technique; consequently, it is referred to as the oculo-basic technique. Examination is directed toward evaluating the coordinating ability between eye movement and the sacroiliac articulations. Each activity must be evaluated separately and found negative, or corrected prior to evaluating for roll.

As an overview of the test for the roll pattern, the patient's hips and knees are flexed in a manner similar to that in pitch, but both knees are rotated to one side; this puts a torsion in the hips, pelvis, and lower back. If an indicator muscle weakens, the roll pattern is positive. In the next step the patient turns his eyes laterally while maintaining the leg position. If the roll pattern is present, there will be a return of strength to the indicator muscle when the eyes are turned right or left or in both directions. The test is repeated with the knees rotated to the opposite side.

Prior to testing as described above, the patient is evaluated for weakening of an indicator muscle with various eye movements. Ocular lock²³ or eyes into distortion (explained later) could interfere with the test.

Correction is directed toward the sacrum. The patient is prone, with the pelvic portion of the table elevated. The sacrum is contacted by the examiner's thumb, and a static challenge is held cephalad while a previously strong indicator muscle is tested; it should not weaken. If it does, there is some other problem in the pelvis which should be evaluated and corrected. If a strong indicator muscle remains strong with the challenge, the patient is asked to turn his eyes to one side, and the muscle is tested again. Weakening of the indicator muscle determines a positive challenge on that side. The opposite side is challenged in a similar manner. Usually only one side will weaken; if both do, the side of greater indicator muscle weakening is the positive side. When the positive side has been determined, the challenge is repeated with the patient maintaining the lateral eye position; the indicator muscle is tested while inspiration or expiration is held. The phase of respiration which abolishes the weakness indicates the direction in which the sacrum is to be moved. If the weak muscle

was eliminated on inspiration, the correction is pressure on the sacrococcygeal articulation in a cephalad and slightly anterior direction while the patient takes a slow, deep inspiration. This is repeated for approximately two minutes, during which time the patient will often feel relaxation in the trunk and in the neck musculature. If the weak muscle resulting from challenge and eye movement is neutralized by expiration, the sacrococcygeal junction is pressed cephalad and somewhat posteriorly on a slow expiration. After the corrective attempt the patient should be re-evaluated in the supine position.

Yaw

Yaw is the rotation of body modules around the vertical axis. A yaw pattern, then, is indicated by head rotation on the shoulder girdle, making one shoulder appear anterior to the other. There are three yaw patterns, described in applied kinesiology as yaw #1, #2, and #3.

Yaw #1 — Occiput. The patient is supine and tested with his knees and pelvis rotated to one side, and his head rotated to the opposite side. Since the knees and pelvis have already been tested individually in rotation during the roll pattern, only head rotation must be tested individually. With the patient's legs and pelvis flat on the table, have him rotate his head to one side and test an indicator muscle for weakening; if present, a cervical subluxation is usually indicated. When the indicator muscle does not weaken when the patient turns his head in either direction, the hips and knees are flexed, with the knees rotated to one side, and the head rotated to the opposite side. Weakening of the indicator muscle indicates a positive yaw #1 pattern. This represents a different type of atlas-occiput fixation than that described under "Fixations" in Volume I. The fixation is on one side only, which is determined by a



17—2. The atlas transverse process is being held while the occiput is challenged forward for yaw #1 fixation.

two-handed challenge. Challenge is accomplished by holding the atlas on the anterior aspect of the transverse process while pressing the occiput anteriorly. It will usually be positive on the side which was superior when the patient's head was turned during the positive test. The occiput is adjusted for a fixation on that side in the usual manner for an occipital fixation.²³

Yaw #2 — Sacral. The patient is examined for yaw #2 in the prone position. The body modules are challenged by placing a DeJarnette block under one shoulder and another under the opposite anterior superior iliac spine of the ilium. This places torsion through the trunk, causing an indicator muscle to weaken if the yaw #2 pattern is present. Both sides are tested, but only one side should be positive. If both sides are positive, some other factor — such as sacral wobble — is probably present; it should be corrected before further evaluation for yaw #2 pattern.

The yaw #2 pattern is corrected by adjusting the sacrum from posterior to anterior on the positive side; after correction, the blocking at the shoulder and pelvis should no longer cause an indicator muscle to weaken.

Yaw #3 — Dorsal Lumbar. The test for the yaw #3 pattern is similar to that for yaw #2. A DeJarnette block is placed under the anterior superior iliac spine of the pelvis, and another under the opposite lower thoracic cage. This appears to put a torsion into the dorsal-lumbar junction, revealing a fixation not apparent with the fixation examination techniques described in Volume I. The fixation is corrected in the same manner described for fixations in that text.

Tilt

The tilt pattern is characterized by the patient's inability to simultaneously laterally flex his head and neck while flexing the knee and hip without an indicator muscle weakening. The disturbance appears to be in the proprioceptors of the muscles or joints of the cervical spine.

The tilt pattern is tested with the patient supine. First it is necessary to have the patient laterally flex his neck and head without any rotation; test an indicator muscle for weakening. If weakening occurs, there is a subluxation or local involvement of the cervical muscles which should be corrected before testing for the tilt pattern. The patient then returns his head and neck to neutral, and one leg is flexed at the hip and knee so that the foot rests on the table. An indicator muscle is again tested; weakening indicates a localized problem — such as in the hip or knee — which should be corrected prior to further testing. With both areas individually testing nega-



17—3. Test for right tilt pattern.



17—4. Correction is made by the patient attempting to laterally flex his head and neck while physician resists the effort.

tively, the two tests are combined to evaluate for the tilt pattern. Care should be taken, when the patient approximates his ear toward his shoulder in lateral flexion, that there is no cervical rotation. The tilt pattern is positive when an indicator muscle weakens with the combined test.

Treatment for the tilt pattern is directed to the cervical area. The physician makes a broad contact on both sides of the head, and the patient attempts to laterally flex his head and cervical spine in the same manner as during the testing procedure. The physician resists the movement, with the patient exerting the maximum effort possible. Care must be taken that the patient does not rotate his head since it is not as effective a treatment; it may also cause cervical subluxations. The patient is then re-tested with the combined hip and knee flexion and lateral cervical flexion to determine if the corrective effort was successful.

Often a patient exhibiting a PRYT fault will have only one involvement in the entire series. Engel⁵ surveyed one hundred patients for PRYT and found thirty-three subjects with no involvement, forty with one fault, and twenty-seven with two or more. The corrections described are very effective in improving an individual's postural status, and they are important in establishing a solid base from which the stomatognathic system can function.

EYES INTO DISTORTION

Much postural distortion of the body is due to improper stimulation of the equilibrium proprioceptors. Most of these major receptors are located within the stomatognathic system. There has already been discussion of how visual righting, labyrinthine, and body-on-head reflexes can be disturbed by dysfunction within the system. Various procedures, such as the PRYT technique, have been developed in applied kinesiology to locate disturbances in the equilibrium system. Much postural distortion is secondary; consequently, treatment directed to many of the articulations of the distortion is ineffective. The distortion is maintained by its muscular support. One would think that the muscles contributing to that support would always test in a predictable manner; that is, when there is a high shoulder on one side the latissimus dorsi of that side would test weak and the opposite one test strong. In a high percentage of cases this is true. Routinely some factor of applied kinesiology diagnosis and treatment returns the muscle to normal strength as observed on manual muscle testing, and the structural misalignment improves. There are those cases that are somewhat enigmatic because the muscle which postural analysis indicates should be weak tests strong. Obviously, the nervous system directs it to be that way. It appears that the structural imbalance and muscular function are adaptations to a remote disturbance.

There is automatic adaptation of eye position to body activity, postural positions, and postural distortions. Parker²⁰ describes two demonstrations of eye position adaptation which correlate with afferent input of the vestibular receptors. As you read this text, rotate your head back and forth at about three cycles per second. As you continue to rotate your head, note that the words remain clear and you can still read. Now hold your head still and move the text back and forth at about the same rate. The words blur and reading becomes impossible. In the first instance, the semi-circular canals signal the oculomotor muscles to rotate your eyes in the direction opposite head rotation. In the second instance, there is no input from the semi-circular canals to help control the oculomotor muscles. Muscular control of the eyes significantly improves when there is communication of head movement from the vestibular mechanism.

The other demonstration of interaction between the vestibular mechanism and the eyes is observed in a newborn infant as an involuntary reflex called "doll eyes." When an infant is tilted backward from an upright position where his gaze is straight ahead, his eyes roll in their sockets to maintain the original

orientation in space. When tilted forward, his eyes continue to maintain the same position. When tilted to the side, the eye toward the side of tilt rolls up and the contralateral one rolls down, again to maintain the same orientation in space. This reflex gradually disappears during the first month as eye orientation becomes increasingly dominated by visual stimuli.

The equilibrium proprioceptors can be grouped in three locations: (1) those from the ear or vestibule, (2) the eye or visual righting reflexes, and (3) the somatic reflexes. Although there has been a considerable amount of research reported about the activity of these mechanisms, much is still unknown about their interactivity. Parker²⁰ reports on several research projects designed for the "space lab" missions in this decade to better understand the vestibular mechanism. These projects are designed around the advantages of studying the mechanism under weightless conditions.

Mechanoreceptors that contribute afferent information for equilibrium integration are located throughout the body in joints, ligaments, and other structures. There has been less study of their contribution to equilibrium than that of the visual and vestibular mechanisms. Most effort has been directed to the upper cervical body-on-head reflexes, or, as some term them, head-on-body reflexes. These are the tonic neck reflexes of the infant which develop to more sophisticated function in the adult.¹⁴ It appears that the organization between the visual righting, vestibular, and various somatic reflexes constantly adapts when there is an unusual or abnormal afferent input from one of the areas. This probably will be better understood when research is completed in the weightless condition of the space lab experiments on the vestibular mechanism.

Goodheart¹² has observed an action which appears to change the adaptive interaction of the equilibrium proprioceptors. His initial observation came from viewing a patient with recurring sciatic neuralgia at the plumb line. When symptomatic, she would deviate with a lateral lean of 1½" to 2". When asymptomatic, her posture would be balanced. On one occasion the patient was off-center about 2" at the plumb line, and Goodheart had her keep her eyes closed so she would be unaware of the plumb line. At the time Goodheart was evaluating neurolinguistics as described by Bandler and Grinder^{1, 2, 15} (see Volume III), where an individual turns his eyes, depending on the manner in which the brain is accessed. When the patient was asked to turn her eyes down and to the left with her eyes still closed, her body centered on the plumb line from the 2"

deviation. When asked to put her eyes up and to the right, the 2" deviation immediately returned. By repeating the eye positions, she continued to balance or deviate, depending on the eye position. When in the balanced position, the sciatic neuralgia "... would diminish to practically a negligible degree."¹¹

Further study of this unique experience revealed that turning the eyes did not relate to right or left brain activity; rather, it revealed that muscles changed their function when a patient placed his eyes into the direction of postural distortion. This has been called "eyes into distortion" (EID). For example, if a patient's head is tilted down on the right and rotated to the right, the EID position is for the eyes to be down and to the right. There are six primary eye positions: (1) eyes down to the right, (2) right, (3) eyes up to the right, (4) eyes up to the left, (5) left, and (6) eyes down and to the left. Occasionally the distortion indicates that the eyes should be placed directly up or directly down. This occurs when the distortion is strictly in the sagittal plane with no rotation.

With the patient's eyes placed into distortion, the results of manual muscle testing often change. In the previous example of the individual with the high shoulder but a strong latissimus dorsi, the muscle would probably weaken with EID. When the eyes are directed away from the distortion, which is the adapted position, the muscle tests strong. It appears that the eyes are gimbaled in their sockets, similar to an infant's eyes as discussed previously, to adapt to afferent input into the equilibrium correlating centers. When the eyes are placed in a position paralleling the major postural distortion, the neurologic organizational changes of adaptation are negated.

Evidence of the stomatognathic system's integrated activity with eye adaptation is revealed by changing the hyoid orientation to parallel eye movement. An individual may test strong in nearly all muscles of the body but still have postural distortions. Placing the eyes into the distortion may cause numerous muscles throughout the body to weaken. If the hyoid bone is put into the same position as EID, the muscles will again test strong; that is, if the eyes are held down and to the right and numerous muscles weaken, holding the hyoid bone down and to the right — together with the EID — negates weakening of the muscles. In this case it is necessary to evaluate the hyoid in the manner previously discussed and make any indicated corrections. There will often be considerable change in the patient's postural orientation, and EID will probably then test negatively. Of course, other components of the stomatognathic system, as well as structural

problems throughout the body, may also contribute to muscle weakening with EID.

Ocular lock and EID appear to be different mechanisms. Ocular lock seems to relate to whether the two sides of the body are functioning well together and to cranial faults. Almost any indicator muscle will weaken in the presence of an ocular lock, whereas weakening of muscles is more selective with EID. Ocular lock does not relate to the hyoid position in the same way as EID. Positive ocular lock is eliminated by correcting switching (see Chapter 18), and muscles testing weak with EID are corrected in the usual AK manner.

EID seems to disrupt the body's compensating mechanisms to reveal muscular imbalance which does not appear in the clear as tested with manual muscle testing. When EID causes a muscle to test weak there may be need for additional stimulation to a reflex, such as the neurolymphatic reflex, even though that stimulation may have been previously provided. In other words, it sometimes gives evidence that more extensive treatment of the type previously rendered is needed.

Sometimes it is difficult to determine the major postural distortion. For example, is a head low and to the right, or high and to the left? Occasionally the problem is determining which body module is the major distortion. The head may be low and to the right and the pelvis low and to the left. Schmitt²¹ found that when the eyes are moved in the direction opposite EID, i.e., "eyes out of distortion" (EOD), there was strengthening of a muscle weak in the clear. This is hypothesized to be due to increasing the compensatory action of the eye position which takes place because of the distortion. This assumes that the EID position takes away the body's pattern of eye compensation for the distortion and is the cause of muscles weakening with the eyes into distortion. Simply stated, EOD improves the body's eye compensation for the distortion, and EID takes away the body's adaptation.

The EOD method to determine the position in which the eyes should turn for EID is best done by using the temporal sphenoidal line indicators to find the muscles probably involved. Schmitt uses the example of TS line indicators for psoas and latissimus dorsi weakness on the left and a pectoralis major (sternal division) weakness on the right. Testing all the muscles yields strength, with the exception of the latissimus dorsi on the left. Using the left latissimus dorsi, which is weak in the clear, the eyes are turned into the various positions until one is found which causes the muscle to strengthen. If the muscle strengthens with the eyes up and to the left but is weak in every other position, it indicates that eye

Stomatognathic System Organization To Total Body

position is the compensatory pattern for improved muscle function (EOD). The direction of EID, then, would be eyes down and to the right, taking the eyes away from the compensatory position. When the eyes are in this EID position, the psoas and pectoralis major (sternal division) weaken, and the latissimus dorsi returns to weakness.

In the example presented, all the muscles exhibited as positive on the TS line changed with the EID pattern; this does not always occur. There may be a local involvement causing a muscle to test weak; thus a muscle may not necessarily be a part of the EID pattern. It may require treatment to the neuromuscular spindle cell, origin and insertion, or

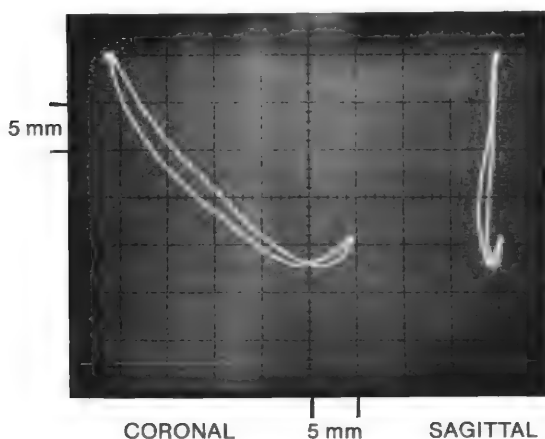
other factor(s).

EID has been a valuable asset in applied kinesiology diagnosis in uncovering subclinical problems. It is often used when a patient has responded well to treatment but reaches a certain plateau where results begin to level out; however, there is still an obvious need for additional correction as observed by the symptomatic complex and the structural distortions present in the body. EID seems to relate especially well with finding additional disturbances in the stomatognathic system that are subclinical and difficult to evaluate, probably because of the strong representation of equilibrium proprioceptors located within the system.

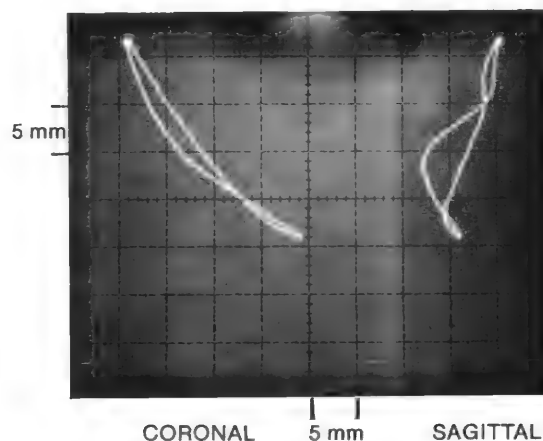
WEIGHT BEARING

The most convenient way in which to examine the stomatognathic system is with the patient supine or reclining in a dental chair. At some point in the examination it is necessary to examine for potential adverse interaction of the stomatognathic system with the rest of the body. Remote dysfunction in the weight-bearing mechanics of the body, the gait mechanism, and general body equilibrium proprioceptors can cause imbalance to return to the stomatognathic system, regardless of how effective corrections may have been.

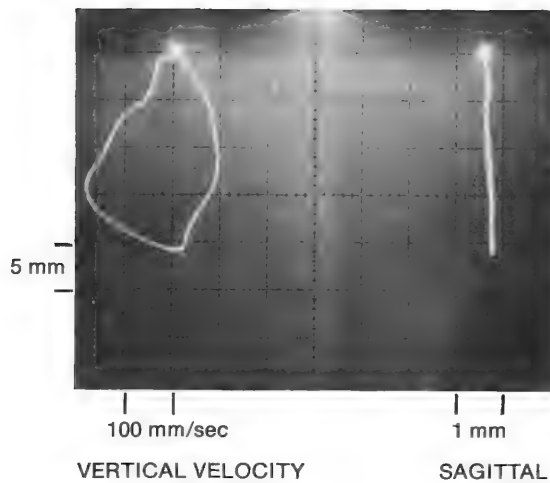
It is very important that there not be improper afferent stimulation into the integrating system from weight-bearing mechanics. McLean et al.¹⁷ demonstrated that the neuromuscular function of jaw closing brought the teeth into the same intercuspa-tion, regardless of extreme position changes from supine toward upright. As described in Chapter 12, there may be change in the occlusion from sitting to standing when there is a weight-bearing problem. In his article considering head posture and its role in occlusion, Mohl¹⁸ states “. . . that studies and



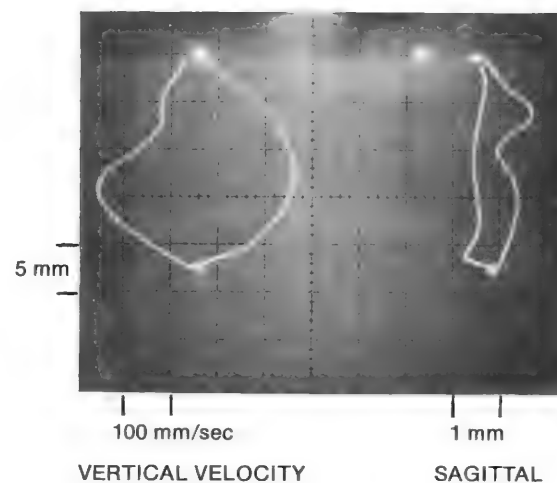
17—5. Mandibular Kinesiograph tracing of mandibular movement in the sagittal and coronal planes when the patient is seated. The bottom curve is an artifact created when the magnet attached to the lower incisor teeth went outside the magnetometer array.



17—6. Same graphing procedure, but the patient is standing. The mandible now deviates to the left 8 mm. A major portion of this patient's problems was found in the feet.



17—7. In the seated position the mandible opens straight. Maximum speed at which the mandible can open is greatly reduced, to 100 mm/sec and 180 mm/sec closing (normal is 300-400 mm/sec). The engram slows movement considerably before reaching intercuspation.



17—8. In the standing position the patient is able to move the mandible faster, but the closing movement becomes erratic. The engram continues to slow movement prior to intercuspation.

consideration of head posture are not merely of academic importance nor an intellectual exercise. It is a factor of clinical significance and should be accounted for in occlusal therapy procedures.”

It has been stressed that leveling the head is a primary correction necessary in balancing activity in the stomatognathic system. This is accomplished by examining and correcting the postural muscles of the stomatognathic system — the sternocleidomastoid, upper trapezius, and deep spinal muscles — if there is a level shoulder girdle. If the head is not level and the shoulder girdle is imbalanced, it is highly probable that there is incoordination of the body modules as discussed in the PRYT technique; correction of this problem will eliminate some weight-bearing problems.

Sometime during the examination it is necessary to evaluate the patient for disturbance in the weight-bearing, gait, and equilibrium mechanisms. The order in which this examination is done usually varies with the physician in charge. When a patient consults a chiropractor for a leg, back, or neck problem, the examination usually begins with the area of complaint. Treatment of the local problem may include corrections to some of the factors discussed in this

chapter. If the chiropractor is knowledgeable about the stomatognathic system, it will probably be examined later in the total procedure unless there is specific body language indicating its primary involvement. Initial examination by a dentist nearly always leads to corrections in the stomatognathic system, followed by examination to determine if remote body function is adversely influencing the system. Since this text is directed primarily to the stomatognathic system, the approach will concentrate on correcting the stomatognathic system, first in the supine position and then by determining if remote factors are adversely influencing it. The methods of examining in the reverse order, where the stomatognathic system is adversely influencing remote factors in the body on an orthopedic basis, are discussed in Volume IV of this series.

After correcting the stomatognathic system as discussed, it is relatively easy to screen an individual for disturbance in the system from remote factors. This is done by examining the patient while he stands and after he walks. Evaluation while standing determines if the patient has a generalized body disturbance from the weight-bearing mechanism. Test a

Stomatognathic System Organization To Total Body

muscle in the supine position to ascertain that it is strong. This muscle will be used as an indicator when testing in the weight-bearing position. The deltoid or latissimus dorsi is good as a general body muscle, and the neck flexors or extensors as muscles relating specifically with the stomatognathic system. After determining that these muscles are strong, or strengthening them with the usual applied kinesiology procedures, have the patient stand and re-test the muscles. If they weaken, there is a general adverse influence on the body from the weight-bearing mechanism, usually from foot, pelvic, or spinal dysfunction. Weakening at this stage requires differential diagnosis to determine what is involved. Examination and correction of the pelvis and spine are discussed in Volume I; the foot and other extraspinal complexes are discussed in Volume IV and other applied kinesiology reference material.^{8, 9, 22}

If there is no weakening of general body muscles when standing, have the patient walk and perform other activities which stress the body in various ways. It is best to have him walk in a figure eight pattern. This stresses the feet, ankles, and pelvis with both right and left turning, as well as with straight walking. Additional stressing can be done by sitting down, standing up, bending over, twisting, etc. None of these activities should cause recurrence of the disturbance just corrected in the stomatognathic system. If they do, differential diagnosis throughout the body should be done just as when there was weakness upon standing. Now additional factors may be involved, such as the gait mechanism, cloacal synchronization, reactive muscles, etc.

Examination of the stomatognathic system after stressing the body can be done with the patient supine, as previously described in the text, or standing. When evaluating the patient standing, it is necessary to have him therapy localize with only one hand at a time on the TMJ, so that the other arm can be used for testing as an indicator muscle. If both hands are used for therapy localization on the TMJ, then the indicator muscle must be the neck flexors or extensors. An alternative is to have the patient stand on one leg and lean against a hi-lo table to test a leg muscle.

When there are positive findings in the stomatognathic system after stressing the body, the same type of examination used in evaluating the integration of the system can be done, as described in Chapter 16. First the stomatognathic system is corrected, and the patient progressively adds different types of stress until the positive findings are again present in the system. Simply have the patient sit up and then test the stomatognathic system. If positive findings are present when the patient is seated but not when

lying down, it indicates that something added by sitting is primary to the stomatognathic dysfunction. The disturbance is probably in the pelvis, spinal column, or equilibrium proprioceptors or cloacal synchronization. Examine the added factors, making corrections where necessary. When the patient can sit without disturbing the stomatognathic system, have him bend into various positions and then test the system again. If positive, the added motion indicates it is probably due to spinal or pelvic fixations which should be evaluated.

When activity can be done in the seated position without adversely affecting the stomatognathic system, standing is added to the progressive testing. An adverse response in the stomatognathic system indicates that the feet and their neurologic positive support mechanism should be tested. The problem will often be found there, but it could be in the knees, hips, or any of the factors considered in the sitting position. The reason the latter may test positive with the patient standing but not sitting is that they may have an adverse interaction with additional factors added when the patient stands. If they are positive standing but not sitting, they are probably secondary to something added when standing. When standing creates no adverse response in the stomatognathic system, the patient bends, adding motion in various directions. This may include squatting, bending to touch the toes, lateral bending, etc. Again because of motion, fixations may be responsible for a positive response, or the stomatognathic system may be reactive to postural muscles anywhere in the body. In problem cases, it may be necessary to have the patient bend in one direction and then test the stomatognathic system; if negative, bend in another direction and test the system, continuing this process until the exact movement causing the adverse response is determined. Then it is easy to individually test the muscles which are activated in that movement to determine the one responsible for the return of the positive stomatognathic system findings.

This type of reactive muscle testing — where one muscle is tested to determine if the stomatognathic system is reactive to it — can very often be done in the prone or supine position. For example, if the patient's positive findings return after touching the toes, some of the extensor muscles of the body are probably reactive to the stomatognathic system. Have the patient lie supine and progressively test the extensor muscles. First the hamstring muscles are tested on one side by having the patient flex at the hip and knee. The physician tests the hamstrings by stabilizing at the knee while extending the leg, with force applied to the posterior ankle. Immediately after this contraction, test the factor in the stomato-

gnathic system which has returned; if negative, test the other hamstring group, gluteus maximus, soleus, gastrocnemius, etc., until a positive finding is observed. Very often the muscle(s) responsible for reactivity in the stomatognathic system can be located in this manner; if not, the patient should be so tested in the standing position. When the muscle is located, it is evaluated for what seems to be an overactive neuromuscular spindle cell. The spindle cell is treated and the test repeated to determine the therapy's effectiveness. (See reactive muscles, Vol. I.)

So far in differentially diagnosing adverse body function on the stomatognathic system, minimal factors have been progressively added while evaluating the system for recurring disturbance. First there

was simply weight bearing in a seated position. Motion was added to the seated position, then static standing was evaluated, and, finally, motion was added to that. Quite often correction of the factors found during these progressive steps completely eliminates any adverse effect on the stomatognathic system from remote body dysfunction.

The next step in the study is to combine these factors and have the patient walk. This tests the body in a much more dynamic manner, and it also evaluates the very important neurologic aspects of the gait mechanism. It is best to have the patient walk in a figure eight; this tests right and left turning, as well as walking straight.

GAIT MECHANISM

The return of disturbance in the stomatognathic system after a person walks or runs indicates probable disturbance in the gait mechanism. The basic approach to treating the gait mechanism is presented in Volume I; it is expanded here to correlate with the postural muscles of the stomatognathic system. A brief review of the gait mechanism and the applied kinesiology approach to examination and treatment is presented first.

There is predictable neurologic control of the muscles as an individual walks and runs. Reciprocal inhibition⁴ is responsible for antagonistic muscles releasing when a prime mover moves a limb. This organization in the gait mechanism can be observed with manual muscle testing by having an individual simulate a gait position. First the subject is tested standing to determine that the shoulder flexors and extensors are normal. He is then put into a simulated gait position, with the right leg forward and carrying most of the weight. In this position a normal individual will test weak in the right shoulder flexors and left shoulder extensors because of the cross pattern movement of the arms when walking or running. It is observed in applied kinesiology that when there is inappropriate stimulation of proprioceptors as a result of foot or other subluxations, the facilitation and inhibition pattern of the shoulders, hips, knees, etc., is random rather than predictable. After the subluxation or other dysfunction is corrected, the predictable pattern of facilitation and inhibition returns. Very often the primary cause of a poor gait pattern is foot dysfunction, which may be a result of subluxations, as mentioned, or active meridian points on the foot which have been related to the gait mechanism in applied kinesiology.

Gait evaluation is done in applied kinesiology by testing two groups of muscles at the same time — groups which function together in normal gait activity. There are six tests consisting of upper and lower muscle groups which are always contralateral.^{3, 10} They are shoulder and hip flexors, shoulder and hip extensors, shoulder and hip abductors, shoulder and hip adductors, gluteus medius and abdominals, and psoas major and pectoralis major.

Activity similar to the facilitation and inhibition of the extremity muscles during gait occurs in the trunk. Activity of the psoas major and pectoralis major muscles applies torsion to the trunk, as does that of the gluteus medius and the abdominals. This torsion through the long axis of the trunk is necessary for balanced movement while walking or running; it is controlled by the same type of inhibition and facilitation of muscles as limb movement. Trunk rotation during gait extends into the relationship of the shoulder girdle, cervical spine, and head. This organization has been described by Goodheart as the "walking gait."¹³

When walking, as the right leg moves forward with hip and knee flexion, the right pelvis moves forward also. Simultaneously the left shoulder flexes. In order for this to occur, there must be inhibition of the left latissimus dorsi and facilitation of the pectoralis major. Manual muscle testing of the subject in this position will confirm a normal weakening of the left latissimus dorsi, while the pectoralis major tests strong. The opposite occurs in the right shoulder; the pectoralis major tests weak because of inhibition, and the latissimus dorsi tests strong. Because of this and other muscle activity,

Stomatognathic System Organization To Total Body

axial torque takes place from the pelvis to the shoulder girdle, moving the right pelvis and left shoulder girdle forward. While the shoulder girdle rotates back and forth during gait, the head remains relatively free of rotation because of the role played by the sternocleidomastoid and upper trapezius in the gait mechanism.

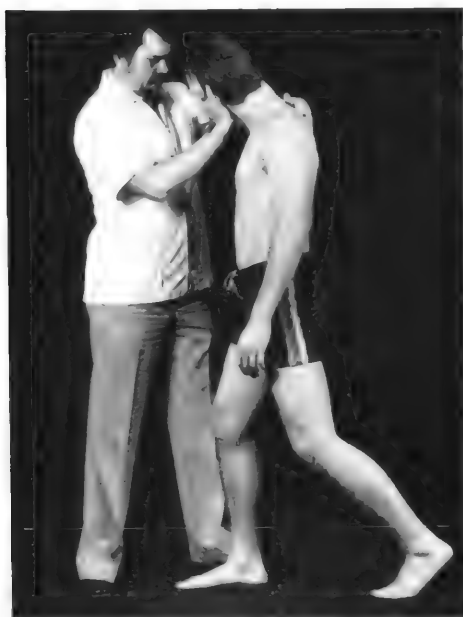
In the same gait position previously described, with the right leg and left arm and shoulder forward, the head must turn to the left in relation to the shoulder girdle to maintain a straightforward position. This is accomplished by facilitation of the upper trapezius on the left and inhibition of the sternocleidomastoid muscle on the left. When a normal subject is in this gait position, the sternocleidomastoid should test weak on the left and strong on the right. In the opposite gait position, the converse is true.

Goodheart¹³ described a lack of this predictable organization especially in individuals with disturbance in the stomatognathic system. His initial investigation included several patients suffering from chronic-clonic-tonic intermittent torticollis. Disturbance in this portion of the gait mechanism relates to recurrent cranial faults, and also to temporoman-

dibular joint and hyoid problems. It can easily be recognized how improper actions of the sternocleidomastoid and upper trapezius on the cranium can disturb its function. Because of the sternocleidomastoid's insertion into the mastoid process, its inappropriate action introduces a force into the cranium's closed kinematic chain very similar to that of the therapeutic force used in applied kinesiology. This force can be detrimental as well as beneficial.

The primary problem with the sternocleidomastoid and upper trapezius muscles in relation to gait is failure of the muscles to be effectively inhibited during the appropriate phase of the gait pattern. This is tested in applied kinesiology with the patient in a gait position to determine if predictable weakening of the muscles occurs. First the sternocleidomastoid and neck extensor muscles are tested with the patient standing in a neutral position to determine that they are strong without gait activity. If there is weakness during this test, correction must be obtained using standard applied kinesiology techniques, described in Volume I and in this text, before the gait test can be done.

These muscles are more difficult to test in a weight-bearing position, and care must be taken that



17—9. To test the sternocleidomastoid in the gait position, the examiner must effectively stabilize the posterior thorax. The sternocleidomastoid should test weak on the trailing leg side.



17—10. Testing the upper trapezius and deep cervical extensors in gait activity is accomplished by the examiner contacting the head to separate it from the shoulder in lateral and anterior flexion. The patient's head is not rotated away from the side of testing as in the standard upper trapezius test. The complex of extensor muscles should test weak on the forward leg side.

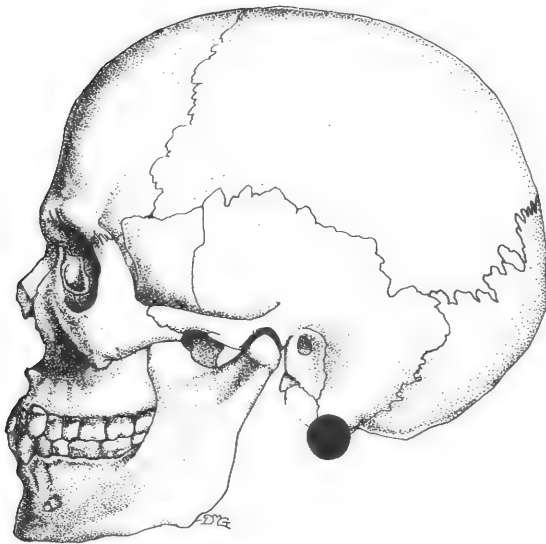
the tests are done accurately. When testing the sternocleidomastoid, the patient should be stabilized with a broad contact of the physician's hand at the upper thoracic spinal area. The extensor test evaluates a portion of the upper trapezius and the deep extensor muscles of the cervical spine. It appears to be a modified upper trapezius test, but is in reality a group muscle test as done in other gait testing procedures discussed in Volume I. The patient's ear and shoulder are approximated as in the standard upper trapezius test, but the patient's head is not rotated away from the side being tested. The examiner stabilizes the shoulder, as in the standard upper trapezius test; his other hand is placed over the head to separate the shoulder and head. The examiner's force is applied in such a manner that the head is brought into lateral and anterior flexion. The usual testing of the neck flexors or extensors in a weight-bearing position — having the patient lean against an upright hi-lo table — is not applicable in this case because the patient must be placed into gait positions.

In the presence of strong muscles, the patient is placed into a gait position with one leg forward, carrying the majority of the weight. The contralateral shoulder is flexed. The contralateral or trailing leg carries a slight amount of weight, primarily on the ball of the foot, with the toes flexed and the heel raised from the floor. When in this position, the sternocleidomastoid should test strong on the side of the forward leg, and the extensor complex should test

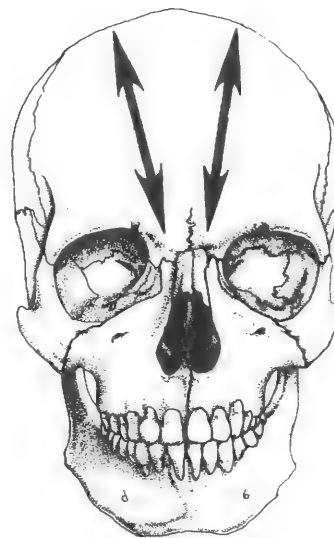
weak. On the opposite (trailing leg) side, the extensor complex should test strong and the sternocleidomastoid weak.

When the muscles fail to weaken at the appropriate time in gait positions, there is usually disturbance in the gait mechanism, such as described previously. There will also probably be an active stress receptor relating with the muscle. The applicable stress receptors are on the anterior lateral portion of the mastoid process for the sternocleidomastoid and on the frontal bone for the upper trapezius. The deep cervical extensors — primarily the splenius capitis and semispinalis capitis — have their stress receptor on the posterolateral aspect of the mastoid process. They are challenged with the patient in the normal standing position to determine the direction of skin manipulation which causes the associated muscle to weaken. The patient is then asked to take a deep breath in, and the muscle is tested to determine if it strengthens; if it does not, the test is repeated with expiration. Usually challenge will be abolished by inspiration. The stress receptor is treated with a heavy manipulation of the tissue in the direction causing muscle weakness, on the phase of respiration that abolished the challenge. This is usually repeated four or five times. The muscle should weaken during the appropriate phase of gait when the patient is re-tested to determine the effectiveness of treatment.

The hip flexors and extensors should have the same type of facilitation and inhibition with gait as do the shoulder and neck muscles. This is more difficult



17—11. *Sternocleidomastoid stress receptor.*



17—12. *Upper trapezius stress receptor.*



17—13. Gait test of rectus femoris. The weight-bearing leg is flexed at the hip and knee to simulate the gait position.



17—14. Psoas gait test requires careful pelvic stabilization by the examiner. The patient can help with the stabilization by holding onto the table.

to test, but a simulated gait test can be done with the patient leaning against an upright hi-lo table. The patient's weight is carried by the forward leg of a gait position, with the foot placed on the forward portion of the foot rest. The hip flexors, such as the psoas and the rectus femoris, should be facilitated. The psoas and rectus femoris of the trailing leg, which is the free leg in this test, should be inhibited and thus test weak. If these muscles fail to weaken on the non-weight-bearing leg, there is evidence of inhibition failure during gait. The stress receptors for the involved muscle are tested in the manner previously described. If the stress receptor is not involved, the problem is usually a reactive muscle; examine with the usual procedure described in Volume I.

The muscles of mastication are also involved with the walking gait. It appears that the jaw-closer muscles are inhibited on the same side as the extensor complex during gait activity. Funakoshi and Amano,⁶ in studying the effects of the tonic neck reflex on the jaw muscles of the rat, found that when the head was turned there was no activity of the masseter, temporal, and digastric muscles contralateral to head turn, but it increased ipsilaterally. There was also bilateral electrical activity of these muscles with forward flexion of the head, but none on tilting. Extension inhibited electrical activity of the muscles.⁷ This activity apparently came from stimulation of proprioceptors in the upper cervical ligaments, since the muscle activity was abolished when the first three cervical nerves were cut.

The criterion that Goodheart¹³ established for testing this factor is to have a patient clench his teeth on the side opposite the normally inhibited trapezius. Clenching the upper and lower opposite quadrants in intercuspation should not neutralize the gait trapezius weakness. It has been clinically found that treatment to the stress receptor for the TMJ muscles on the side opposite clenching eliminates the negation of the upper trapezius inhibition on contralateral clenching. This seems to indicate that there is a reciprocal inhibition between the right and left jaw-closing muscles during walking.

Gait Exercise

Goodheart¹³ has devised a follow-up exercise for the walking gait problem. It has been especially valuable for patients with chronic-clonic-tonic intermittent torticollis.

The exercise is designed to increase the patient's gait activity on the side functioning effectively. The patient is evaluated as previously described to determine which muscles fail to release normally. It is presumed that the opposite side is the one which functions normally, which can be clinically confirmed by watching the patient walk. The head should maintain its forward position during shoulder girdle rotation. When the normally functioning side is in the stance phase of gait, the head will be straightforward. The head and neck tend to pull from the centered position when that leg is in the swing phase of gait and the other leg is in the stance phase. The exercise procedure Goodheart devised is for the patient to take a full step with the leg on the side of normal function, and then to follow with a half-step with the leg on the side of abnormal function. This is repeated several times throughout the day. The rationale for

this exercise is to neurologically pattern the individual toward the normally functioning side. This is the side that goes through the full phase of gait, while the side that is not functioning as efficiently goes through only a half-phase of gait. The number of repetitions depends upon the severity of the problem. In chronic-clonic-tonic intermittent torticollis cases, he has the patient perform the procedure for five minutes every hour during the day; in very severe cases, it may be increased to fifteen minutes every hour for several days during the initial treatment.

It is particularly important to test the gait muscles for normal facilitation and inhibition in the various gait positions when a condition recurs after walking. It is not uncommon for a patient to walk his problem right back in, perhaps even before reaching his automobile to drive home after treatment.

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Chapter 18

Relation of the Stomatognathic System With Various Health Problems

Throughout this text there have been many references to dysfunction in the stomatognathic system causing health problems throughout the body. This is particularly evident in the disturbance of cranial nerve function as a result of apparent peripheral nerve entrapment. This chapter briefly summarizes the role played by the stomatognathic system in various types of health problems. The discussion will certainly not be all-inclusive, since it appears that dysfunction in the stomatognathic system can, either directly or indirectly, relate with the majority of health problems. These brief summaries of various conditions are for association purposes only. The major discussion on applied kinesiology evaluation and treatment of the various conditions is in Volumes I and especially IV and V of this series.

When examination leads to treatment in the stomatognathic system, many ramifications can develop. The system may be entered primarily to relieve chronic headaches or other conditions, and side benefits result from correction of numerous health problems which have not been examined by a physician. The patient may not have felt the problems were serious enough to consult a doctor, or they have been managed by over-the-counter medications. A physician may never be aware that treatment to the stomatognathic system has correct-

ed other problems if he fails to do a thorough case history and read body language. In fact, a patient may not recognize the benefits, thinking that the sinus drainage, neck pain, or ringing in the ears simply went away by itself.

When body language, which has been described throughout this text, indicates dysfunction of various components of the stomatognathic system, a physician should question the patient about symptoms relating to the observed findings. Considerable additional information which the patient may not think important enough to mention will often be added to the case history. Skill in reading body language and thus bringing out additional problems secondary to the primary dysfunction will help a patient better understand the integration of body activity. The physician who is a neophyte in applied kinesiology will soon recognize the wide range of conditions applicable to treatment as determined by AK examination.

A comprehensive discussion is included in this chapter about locating the cause of neurologic disorganization, which has been called "switching" in applied kinesiology. Although neurologic disorganization is presented in Volume I, it is not discussed thoroughly because of the student's lack of knowledge about the stomatognathic system, which plays an important role in this type of disorganization.

Relation of the Stomatognathic System With Various Health Problems

Conditions

Allergies

Natural health treatment reveals that often when an individual is sensitive to a particular substance and is classified as allergic, it is because tissue health is lowered and cannot effectively cope with an irritant as it does in normal health. Rhinitis and conjunctivitis are very often improved or corrected by balancing the stomatognathic system. This appears to be due to improved cranial nerve function which controls secretions in the eyes and nose. Rhinitis may be due to vasomotor disturbances without an allergic basis and improve in a similar manner.

Allergic gastroenteritis may be secondary to improper vagus nerve (X) control of the blood vessels of the gut, mucous, and hydrochloric acid secretion. Hypo- or achlorhydria can cause failure of normal protein digestion to which the gut may be sensitive. Under these circumstances calcium is poorly absorbed. A treatment used in the 1920s and 30s was injected calcium, which reduced allergic reactions but failed to provide any lasting effect when the injections were discontinued.²¹ The working hypothesis in applied kinesiology that is clinically effective for allergies is to improve hydrochloric acid secretion with cranial fault correction, which in turn improves calcium absorption. Clinical evidence shows entrapment of cranial nerve X, resulting in failure to control the secretion of hydrochloric acid and mucous, and probably improper control of blood circulation to the gut.

In addition to those mentioned, other types of allergies often improve with correction of the stomatognathic system. Besides the working hypothesis mentioned, improved adrenal function and other factors probably contribute to the relief of an apparent allergic reaction.

Auditory

There has been much discussion in the literature indicting the stomatognathic system as a cause of various types of auditory problems. Prompting much of the discussion was Costen's⁷ strong indictment of the temporomandibular joint and occlusion as a major cause for lesions of the sinuses or ears. Although his hypothesis of the etiology of auditory symptoms has not stood the test of time, the association of symptoms improving after balancing the stomatognathic system has. Common embryologic development of the auditory mechanism and stomatognathic area seems to offer an understanding of the association of auditory problems with disturbance in the stomatognathic system. The tensor tympani receives a nerve supply common with the internal pterygoid muscle.² The tensor palati opens

the eustachian tube; it is also innervated by cranial nerve V.³ It appears that either muscular dysfunction or cranial faults can disturb these nerves.

There is a direct mechanical relation of the temporomandibular joint with the middle ear, not generally discussed in standard anatomy texts.^{17, 38} The connecting structure is a tiny ligament, discovered by Pinto,²⁸ which connects the neck and anterior process of the malleus to the medioposterosuperior part of the capsule, the interarticular disc, and the sphenomandibular ligament. The ligament is called the mandibular-malleolar ligament⁹ and is described on page 300.

Narrowing of the eustachian tube has been associated with hearing losses at the 6,000 cycle level.⁵ This may be due to the muscular disturbance described above or allergic reaction, either of which can be the result of dysfunction in the stomatognathic system.

Barnard⁴ relates some impediment to speech clarity to a transitory hearing loss attributed to TMJ problems. It is difficult to determine whether the speech problem results from an auditory disturbance which may be present, or from other factors disturbed because the general stomatognathic system is out of balance. There may be a disturbance of the cranial nerves involved in phonation as a result of the general problem.

When there is disturbance in auditory function, special examinations are required to differentially diagnose the problem. It is valuable for those working with the stomatognathic system to at least have the equipment to do auditory screening tests. Gelb et al.¹³ point out the importance of dentists and otolaryngologists being aware of each others' specialty areas and the interaction between them. This is true of all the specialties working with the broad scope of the stomatognathic system.

Vertigo

There are many reasons the stomatognathic system should be thoroughly examined when an individual has vertigo or other equilibrium problems. There may be peripheral entrapment of the vestibular division of cranial nerve VIII,²³ or there may be disturbance in the relationship of the right and left vestibular mechanism as a result of counter-rotation of the temporal bones.²⁶ This type of counter-rotation is caused by faults such as a temporal bulge on one side with a parietal descent on the other, or inspiration and expiration assists on opposite sides.

Any disturbance within the stomatognathic system which interferes with the equilibrium propriocep-

Relation of the Stomatognathic System With Various Health Problems

tors can cause vertigo or general equilibrium problems. The stomatognathic system as a cause of vertigo should be differentially diagnosed from various types of upper motor neuron lesions. Vertigo from cervical involvement is relatively common. Tests for its differentiation from otolith vertigo have been developed⁶ in which an individual is stabilized with a collar which steadies the head, neck, and shoulder girdle. The patient, thus stabilized, is lowered from a sitting to a prone position. If the patient develops no vertigo with the collar but does when lowered without it, there is evidence of cervical vertigo as opposed to otolith vertigo. This procedure is seldom required when applied kinesiology challenge and therapy localization are used for differential diagnosis. Diagnosis of neurologic disorganization, often the etiology for vertigo, is discussed more thoroughly later in this chapter.

Paranasal Sinuses

It is not precisely clear why many sinus conditions improve with correction of the stomatognathic system. Although Costen⁷ described both sinus symptoms and auditory problems as occurring from disturbed function of the temporomandibular joint, he made no particular effort to explain the mode of the sinus disturbance as he did with the auditory portion; yet some of the case histories he reported referred to improvement of sinus conditions and rhinitis. In light of today's understanding of the stomatognathic system, it appears that these conditions — along with headaches — were probably corrected by a combination of factors. Study of a disarticulated skull reveals very thin bone structure in many areas of the paranasal sinuses. In classic studies, Wolff⁸ has described pain sensitivity and localized referred pain from stimulation of the paranasal sinuses. Cranial corrections often provide immediate relief from pain attributed to sinus symptoms but which may be due to mechanical pressure on the sutures. Retzlaff et al.^{29, 30} described nerve endings within the sutures where pressure could be relieved with this type of manipulation. Magoun²⁴ describes structural distortion from cranial faults as interfering with adequate circulation of blood, lymph, and air to this area. Correction of cranial faults often results in improved sinus drainage shortly after treatment.

The role of the stomatognathic system in allergic reactions may also influence certain types of sinus conditions. Probably much relief of headache and facial pain attributed to improved sinus function is, in reality, a reduction of referred pain from other areas in the stomatognathic system, such as from muscular imbalance causing referred pain from trigger points.

This type of pain is often falsely attributed to the sinuses by patients and physicians alike.

Mental Health

There are certain types of mental health problems which respond uniquely well to applied kinesiology evaluation and treatment. Much of the treatment necessary in correcting mental health problems is directed toward the stomatognathic system. Woods and Woods³⁹ found in a study of 102 psychiatric patients that the average cycling of the cranial primary respiratory pulse was 6.7 cycles per minute, while in a control group of sixty-two normal subjects the average was 12.47 cycles per minute. Fonder and Allemand¹⁰ studied two groups of children in a summer academic program. One group was remedial, comprising children attempting to catch up with their studies and pass on to the next grade; the other group was for the enrichment of students excelling in their studies. In the remedial group, 100% had malocclusion and 83% had severe malocclusion; only 2% did in the enrichment group. The remedial group had 31.9% with serious psychological problems, whereas in the enrichment group there were no serious psychological problems.

One type of mental health problem often corrected by applied kinesiology treatment directed to the stomatognathic system is neurologic disorganization characterized by a homolateral crawl pattern. Normally an individual's nervous system is enhanced by a cross crawl pattern and disturbed by a homolateral crawl pattern, which occurs when ipsilateral upper and lower extremities move in flexion simultaneously. The normal cross crawl pattern is contralateral extremities moving together in extension or flexion while walking or crawling. Goodheart¹⁵ recognized that schizophrenic patients consistently have a homolateral crawl pattern. This author reported a method for changing the homolateral crawl pattern to the normal cross crawl pattern to members of the International College of Applied Kinesiology.³⁶ The approach has been successful in treating schizophrenics, although results are limited for those who are on heavy medication. (Examination and treatment of mental conditions are discussed in Volume V.)

Learning Disabilities

Many types of learning disabilities can be improved with treatment to the stomatognathic system. Upledger³⁵ reports a statistically significant lack of cranial-sacral motion in children who are classified by school authorities as "learning disabled." The study by Fonder and Allemand,¹⁰ noted above, found that in addition to 100% having dental malocclusion, 100%

Relation of the Stomatognathic System With Various Health Problems

had a loss of hearing acuity, with 45% having over 20% loss. Forty-five percent complained of more than one-third of the health problems associated with dental malocclusion.¹¹ Frymann¹² points out the importance of treating the cranial-sacral primary respiratory mechanism in the early critical years of life, but she also recognizes the value of treatment for the older student. Applied kinesiology experience concurs with this. When a child goes through the early grades with a handicap of neurologic disorganization, visual disturbances, auditory problems, etc., it is difficult for him to catch up in later years, even if adequate correction is relatively easily obtained.

As will be discussed later in this chapter, neurologic disorganization is often due to inappropriate stimulation of nerve receptors within the system which give conflicting afferent information, causing generalized confusion within the system. This confusion may be responsible for conditions such as dyslexia, where an individual interprets visual and vocal communication in a confused manner. These individuals may have ocular lock, diplopia, visual and auditory disturbances, and many other conditions relating directly to dysfunction in the stomatognathic system.

Headaches

Accurate diagnosis of headache etiology is often challenging. Although the primary cause of a headache may be remote from the pain, the stomatognathic system is nearly always involved on at least a secondary basis. The correlation of suture jamming, trigger points, local muscle pain, cranial nerve entrapment, and TMJ dysfunction with headaches is obvious.

It is not uncommon to initially diagnose a remote cause for a headache, such as a digestive disturbance causing a toxic vascular headache, only to find on further investigation that dysfunction in the stomatognathic system is the primary cause. This could result from cranial nerve X entrapment by cranial faults, or muscle and fascia entrapment as the nerve courses through and between these structures. The pH of the bowel may be disturbed and enzymes released inappropriately, along with improper bowel motility, finally resulting in an ileocecal valve syndrome. Symptoms may be wide-ranging and include headaches. Here the primary disturbance is within the stomatognathic system, but initially it may not cause a headache. It is only after toxicity from the resulting digestive problem causes a vascular spasm that the headache occurs. Appropriate diagnosis and correction of this condition require that a physician be familiar with the chain of events which can take place when the stomatognathic system creates a

remote problem, in turn causing symptoms related back to the stomatognathic system.

Gelb and Tarte¹⁴ recommend a coordinated effort among the various professions of the healing arts in dealing with the problem of headaches. Too often the occlusion and temporomandibular joint are not considered in cases of chronic, severe headache. The muscles and fascia of the postural portion of the stomatognathic system, along with vertebral subluxations, must also be considered as a possible etiology. Page²⁷ discusses these structures as a cause of headache, along with some of the neurology of head pain. It is important that the total stomatognathic system be evaluated for function, and an individual should be studied for possible pathological causes. Today the trend seems to be toward more and more use of sophisticated equipment, such as a CAT scan, which is valuable for differentially diagnosing pathologic conditions. Unfortunately, the integrated function of the structures involved is too often ignored. A balance between searching for pathology and evaluating function must be developed for the optimum differential diagnosis of the cause of headaches.

Referred Pain

There has been considerable mapping of referred pain, which may be from the teeth to various areas of the head,³¹ or from trigger points.^{33, 34} Although reference charts are valuable in helping to quickly find a remote cause of pain, it is well to keep in mind that the potential pathways of reference are extremely complex and can vary tremendously. Green et al.¹⁸ demonstrated in decerebrate and decerebellate cats that stimulation of any division of the trigeminal nerve induces efferent volleys in cranial nerves VII, X, XI, and XII. Pain may even be referred to the facial tissues by coronary heart disease.¹ When the usual symptoms of coronary pain are present, along with exacerbation by physical activity, eating, emotional stress, etc., differential diagnosis is not difficult. In the absence of these usual signs, the problem of differential diagnosis is compounded. Any condition of the stomatognathic system that is considered functional but fails to respond, both subjectively and objectively, within a relatively short period of time requires further investigation. Understanding of the interaction of the nervous system, such as that described above, indicates additional areas to examine for possible referred pain.

Endocrine Disorders

Any imbalance in the endocrine system indicates a need for evaluation of the stomatognathic system. As always, the primary disturbance may be found anywhere in the stomatognathic system, but the

major cause of endocrine problems seems usually to come from improper cranial primary respiratory function. Magoun²⁵ hypothesizes several ways the pituitary may fail to function normally because of cranial faults. He discusses circulatory restriction, peripheral nerve entrapment, and restricted cerebrospinal fluid flow as possible causes of pituitary dysfunction. Another widely held cranial concept in pituitary dysfunctions is that there is a need for motion of the sella turcica to maintain activity of the pituitary body. The vagus nerve supplies the thyroid,²² which may dysfunction as a result of cranial faults.

Improper function of one of the endocrine glands may begin a chain effect, causing considerable imbalance within the system. There are several methods in applied kinesiology for examining the interaction of the endocrine system. (The endocrine system is discussed thoroughly in Volume V.) One is two-handed therapy localization, described in Volume I. Correction of this interacting functional disturbance recognized in applied kinesiology produces good clinical results. When working with this type of functional condition, it must be explained to the patient that the gland is not classified as diseased; rather, it appears not to be functioning in an optimum manner. These functional disturbances are recognized by those who use natural approaches to regain health, but they may not be recognized as abnormalities by specialists in endocrine disease.

Trauma

It seems superfluous to list trauma here as a condition correlating with the stomatognathic system. It is done to specifically point out the importance of evaluating this system when there is a hyperflexion-hyperextension cervical strain or sprain. This condition, often called "whiplash," nearly always includes trauma to other components of the stomatognathic system in addition to the cervical spine. Many patients seen by applied kinesiologists have previously consulted and been treated by numerous physicians, but they have not responded to the therapy provided; in fact, sometimes therapy such as traction, which applies forces into the temporomandibular joint, occlusion, and cranium, often makes the condition worse. The hyoid component is almost always involved, as are cranial faults and imbalance in the postural muscles of the stomatognathic system.

Some of the symptoms which patients attribute to an auto accident or other personal injury are discounted by a treating physician because of his inability to correlate the symptoms with the trauma. An understanding of the stomatognathic system and an ability to easily evaluate it tie symptoms of nausea, vertigo, diplopia, auditory problems, etc., together. Not only does an understanding of the condition develop, but treatment is usually effective, even in severe, long-standing conditions.

Locating the Cause of Neurologic Disorganization

Many health problems relate directly to neurologic disorganization. There are specific predictable patterns which should be present in the nervous system that are often disturbed because of various health problems. In Volume I some types of neurologic organization are discussed, such as bilateral brain function and cross patterning as observed in the gait mechanism. Neurologic disorganization — called "switching" in applied kinesiology — is discussed on a limited basis. Sometimes the nervous system appears to be "confused." This is recognized during manual muscle testing as lack of facilitation or inhibition at appropriate times, when muscle function fails to be compatible with the status of other conditions found in the body. In the past, switching was considered when the manual muscle test findings appeared confused. As more knowledge has been gained in applied kinesiology, neurologic disorganization can be divided into two categories —

predictable and unpredictable. Predictable neurologic disorganization refers to normal or abnormal disorganization, of which the cause is known. Normal disorganization is temporary and benefits the body. An example is excessive energy held in the lung meridian to help combat bronchitis. Abnormal predictable neurologic disorganization occurs when there is a recognizable condition and an effective therapeutic approach is devised to correct it. In the early stages of applied kinesiology, most switching was unpredictable. With increased knowledge, nearly all switching can be put into the predictable category, and a cause for the condition can be found.

An example of what would have been considered confused earlier in applied kinesiology development, but is now considered predictable, is the walking gait mechanism, discussed in Chapter 17. During gait, when the right foot is forward the left sternocleidomastoid should be inhibited and test weak. If it does

Relation of the Stomatognathic System With Various Health Problems

not, it could be classified as a non-predictable finding and therefore switching. With today's AK knowledge, there is a predictable dysfunction of the stress receptor, and probably some problem in the gait mechanism, which are examined for and treated. With the return of normal inhibition, there is predictable function.

Several years ago, Goodheart¹⁵ observed that when unpredictable activity occurred within the body there was consistently a positive therapy localization at acupuncture point K27, located at the junction of the clavicle, sternum, and 1st rib. Simultaneous stimulation of K27 and the umbilicus would very often change the unpredictable findings and organize them. Other types of evaluation and treatment for switching were developed and are discussed in Volume I. When this type of treatment is provided to eliminate evidence of switching, some patients do not return to a switched status; others continually do, and the same stimulation is required on each office visit. It has become obvious that "problem patients" are very often those who continually exhibit switching.

The next observation which began to clarify the switching problem was that when treatment was provided which improved the symptomatic pattern, recurrent switching was also eliminated. It seemed apparent that switching and the primary cause of the patient's condition were related. The logical approach, then, was to find the cause of switching; its elimination was a step in the right direction toward correcting the patient's problem. A system using positive therapy localization to K27 was developed to find the cause of switching; it was reported to the members of the ICAK in 1982³⁷ and is expanded here.

Positive therapy localization at K27 had always been considered an examination finding which should be eliminated. As stated, this was done with simultaneous stimulation to K27 and the umbilicus. At times a point called auxiliary K27, adjacent to T11, was simultaneously stimulated with the umbilicus. The concept of eliminating positive therapy localization to K27 is maintained, but the mode of accomplishment has changed. The approach is to use the positive K27 therapy localization as an examination asset to determine the dysfunction causing the problem. The role of K27 as an indicator in switching is discussed more thoroughly in Volume III. For now it is adequate to consider it an important acupuncture point which becomes active when there is confusion within the controlling systems of the body.

A patient exhibits a positive K27 whenever it is therapy localized and a strong indicator muscle weakens on testing. This is called a "positive K27 in

the clear." A somewhat more revealing situation occurs when a patient shows a positive K27 only under certain circumstances. For example, an individual may not have evidence of a positive K27 when supine; when he stands, therapy localization to the point becomes positive. This is termed a "hidden positive K27."

During the discussion in Chapter 10 of the interdependence of the components making up the stomatognathic system, there is some mention of receptors and effects; it will be briefly repeated here as it relates with the total body and neurologic disorganization. Input to the organizational systems of the body is through receptors, which are classified as mechanoreceptors, thermoreceptors, nociceptors, electromagnetic receptors, and chemoreceptors.¹⁹ In addition to the usual receptors found in physiology textbooks, we will include in our discussion mental receptors, and acupuncture points as a type of electromagnetic receptor. Stimulation of the receptors is the method used to determine the body's status, regardless of whether it is structural, chemical, or mental. Inappropriate stimulation to a receptor puts incorrect information into the organizational systems, which can act only on their perception of what is occurring in the body.

An example of how confusion can develop within the body is provided by a foot condition, such as pronation or tarsal tunnel syndrome. Probably included in the foot condition are subluxations and hyper- or hypotonic muscles. Mechanoreceptors include neuromuscular spindle cells and Golgi tendon organs in the muscles, and free nerve endings in the skin and deeper tissues, as well as joint and many other proprioceptors. When a foot functions normally, these mechanoreceptors are stimulated in an organized way, providing the central nervous system with information about the exact status of weight bearing, movement during gait, etc. With foot dysfunction, the strain on both the intrinsic and extrinsic muscles changes from normal, as does the stimulation to the joint proprioceptors during motion. One or more of the foot muscles may be stretched in such a way that the neuromuscular spindle cell sends afferent impulses, indicating toe flexion is taking place. If there is an extension subluxation of the toe, its joint proprioceptors send extension information. The information received by the central nervous system must be acted upon in order to coordinate inhibition and facilitation of the muscles throughout the body for weight bearing and gait activity. Confusion results, and the central nervous system cannot adequately process the information to control flexion and inhibition of muscles throughout the body. This appears to be the

Relation of the Stomatognathic System With Various Health Problems

reason why people who are switched may exhibit a strong muscle when the body activity indicates that it should be inhibited, as in the example of the sternocleidomastoid in the walking gait previously.

Under these circumstances positive K27 therapy localization is consistently found; however, it may not be present in the clear.

DYNAMIC USE OF K27 AS AN INDICATOR FOR SWITCHING

In Volume I the primary approach to switching is simultaneous stimulation of K27 and umbilicus, connecting the governing and conception vessels, and possibly stimulating umbilicus and auxiliary K27. These approaches organize the systems and appear to override evidence of disorganization. This has a certain value in an examination because it organizes inappropriate findings. Without this organization, a physician might see evidence of a right subluxation when, in reality, it is left. The switching "treatment" organizes these findings so that inappropriate adjustments and other treatment are avoided. This is a basic approach which has been used in applied kinesiology, but there is a better method for dealing with switching as a physician becomes more proficient in AK diagnosis.

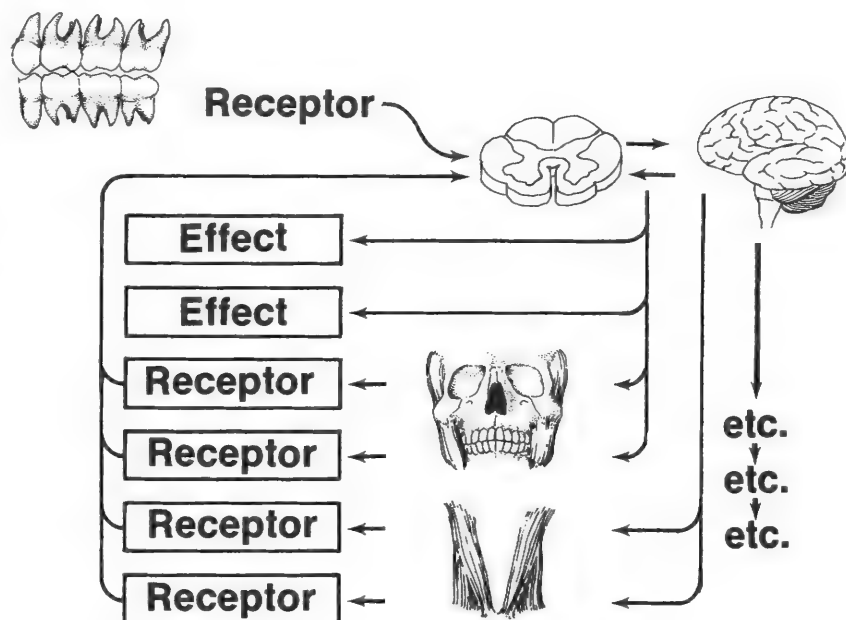
Routine stimulation of K27-umbilicus clears the evidence of neurologic disorganization. This is valuable, as described previously, in preventing improper treatment; however, it is also detrimental in finding basic underlying causes of conditions. Overriding switching eliminates indicators of some conditions which must be treated to eliminate neurologic disorganization. For this reason, routine stimulation of K27-umbilicus is self-defeating. (Examples of hidden primary problems will be presented as this subject is developed.)

The dynamic approach to switching uses positive therapy localization to K27 as an asset in locating the basic underlying cause of the switched condition. This is accomplished with various examination procedures which are used to find what cancels the positive therapy localization. That factor is then treated, eliminating the positive K27; this is a more effective method of abolishing the return of switching.

It is important to understand the effect of simply stimulating K27-umbilicus and connecting the

conception and governing vessels. It does not appear that these methods alone provide effective treatment. They appear to only temporarily organize body function. Evidence of switching will immediately return if no other treatment is given. This can be observed in nearly all instances by the recurrence of switching immediately after a patient walks, chews, stands, or stresses his body in some manner. Recurrence could probably be demonstrated in all cases if the physician knows how to stress the body so that the original cause of the switching — whether structural, chemical, or mental — is stressed.

The consistent return of switching when no other treatment is given indicates that when switching does not return, it is because the physician makes some correction which was responsible for the switching in the first place. Rather than accidentally making the correction, the dynamic method of locating the cause of switching is purposeful and more rapidly finds the basic underlying causes of an individual's health problem.



18—1. When the cause of neurologic disorganization is found and corrected, there will be improved function of the previously improper effects. (See page 284 for more discussion of this model.)

CORRECTION OF SWITCHING IN THE CLEAR

Each new patient should be routinely tested for positive therapy localization to K27. If present, the physician uses the diagnostic tools of challenge, therapy localization, oral chemical challenge, and muscle testing to find what abolishes the positive K27. This factor is then correlated with the usual diagnostic signs of palpation, structural analysis, etc., and perhaps laboratory tests in the case of oral chemical challenge. Treatment is directed toward the factor eliminating the positive K27 therapy localization; if effective, there will no longer be evidence of switching. In this manner K27 serves as a monitor for switching or neurologic disorganization rather than a treatment point. The examination approach to find what eliminates the positive K27 can be divided into the triad of health factors — structural, chemical, and mental. The most common causes of switching are found in the structural group, with a descending frequency in chemical and mental.

Structural

If a patient demonstrates a positive K27 therapy localization when supine, the examiner simply challenges various factors or uses two-handed therapy localization to determine what eliminates it. Body language can often help the physician rapidly find the cause. For example, a patient may have symptoms which increase with daily activity. The examiner's questions reveal that the symptoms develop more when standing and walking, and are relieved when sitting. The indication is structural, probably in the feet or gait mechanism. If a patient indicates progressive symptoms all day which are alleviated when lying down, there is probable pelvic or spinal involvement. The feet and gait mechanisms are probably not involved if relief is not obtained when sitting. In these examples, positive K27 therapy localization will be eliminated when the correct challenge is done by the examiner. If the foot is involved, challenging it in the direction of correction will cause an indicator muscle weakened by therapy localization to K27 to strengthen. Correction of that subluxation will eliminate the positive K27.

A very common cause of switching is dysfunction in the stomatognathic system. Clinical experience indicates that the most common cause of switching is in the stomatognathic system; the second most common cause is in the feet and gait. The high incidence from the stomatognathic system is probably due to its intricate relation with the equilibrium proprioceptors. When body language indicates involvement of the stomatognathic system, as presented throughout this text, the various types of examination

are applied to the cranium, temporomandibular joint, cervical spine, hyoid muscle balance, etc., to find what eliminates the positive K27 therapy localization. If ocular lock is part of the switching pattern, its cause will nearly always be in the cranial primary respiratory mechanism.

A sequential example of locating the cause of switching in the stomatognathic system will help clarify this approach. A patient has a positive K27, indicated by therapy localizing to K27 with the pectoralis major (sternal division), used as an indicator muscle, weakening. The patient's major complaint is suboccipital headaches. To thoroughly explain the role of dynamic evaluation for switching, this discussion will include the results of various examination procedures with and without therapy localization to K27. Vertebral challenge of the atlas without therapy localization to K27 causes an indicator muscle to weaken, indicating a subluxation. When K27 is therapy localized and an indicator muscle weakens, the atlas challenge does not cause the muscle to regain strength, indicating that the atlas subluxation is not part of the switching complex. This is verified by a two-handed therapy localization, with one hand on the bilateral K27 and the other over the atlas area. There is no strengthening of the indicator muscle, which in this case is the tensor fascia lata. Another finding is a right sternocleidomastoid weak in the clear that strengthens when K27 is therapy localized. Testing for improvement of the sternocleidomastoid by therapy localizing the neurolymphatic and neurovascular reflexes, stomach alarm point or upper cervical vertebrae, and having the patient chew niacin and vitamin B₆ does not strengthen the muscle or abolish the positive K27 therapy localization. This indicates that the sternocleidomastoid, although weak, is not responsible for the switching complex. The cranium is challenged with various vectors to determine if the positive therapy localization to K27 is eliminated; it is not. Two-handed therapy localization is applied to K27 and to the temporomandibular joint on the right and then on the left; again, no cancellation of the positive K27 therapy localization is observed. The hyoid is challenged by a support person in various directions and held with simultaneous therapy localization to K27. When challenged right and anteriorly, the indicator muscle — previously weakened from K27 therapy localization — regains strength, indicating an involvement of either the stylohyoid or the posterior belly of the digastric muscle on the left, since those are the muscles stretched with this challenge. Therapy localization and palpation are used to

evaluate the muscles; apparent neuromuscular spindle cell dysfunction is found in the posterior belly of the digastric and corrected. There is now no positive therapy localization to K27 in the clear. The sternocleidomastoid tests strong and the atlas, which previously exhibited positive therapy localization and challenge, is re-tested; it does not now exhibit evidence of a subluxation.

The findings described above are typical of the interaction occurring when apparent neurologic disorganization is present. The atlas subluxation and right sternocleidomastoid muscle weakness are secondary to the apparent dysfunction of a neuromuscular spindle cell in the posterior belly of the digastric muscle. Correcting its improper afferent signaling into the integrating system eliminates the inappropriate sternocleidomastoid weakness, which is probably responsible for the atlas subluxation. Adjustment of the atlas or treatment directed toward the sternocleidomastoid is treating secondary effects rather than the basic underlying cause of the problem.

Another important point can be illustrated in this example. As previously mentioned, many clinical investigations have been done to determine the possibility of treating the wrong factor when treatment is provided prior to eliminating switching. In this example, if K27-umbilicus had been stimulated without investigating to find what abolished the positive therapy localization, the results would have been elimination of the evidence of the atlas subluxation and a strengthening of the sternocleidomastoid. Confirmation that this would occur is the strengthening of the sternocleidomastoid and the elimination of positive therapy localization to the atlas by therapy localizing to K27. Thus stimulation of K27-umbilicus is of value to the neophyte in applied kinesiology because it eliminates indications which may cause him to treat the secondary effects of subluxation and muscle weakness incorrectly. The problem is that he may not find the basic underlying condition; as soon as the patient turns his neck and significantly activates the posterior belly of the digastric muscle, evidence of switching, atlas subluxation, and sternocleidomastoid muscle weakness will return.

FINDING AND CORRECTING A HIDDEN POSITIVE K27

As a diagnostic tool, K27 is even more valuable when it is negative to therapy localization in the clear. Almost all — if not all — patients with a chronic problem, or those who do not respond adequately, have neurologic disorganization which can be recog-

Chemical

Switching may be caused by chemicals when some biochemical or toxin stimulates the chemoreceptors in such a manner that confusion results within the system. Chemical causes of switching can be evaluated in a manner similar to that for structural causes. Body language again indicates the possible presence of a chemical factor. If a patient has exacerbation of symptoms on a monthly cycle with menstruation or ovulation, hormone balance is suspect. This can be evaluated by having the patient chew nutritional substances that may relate with the endocrine imbalance, or by electron poisoning evaluation. These subjects are discussed in Volume V of this series and have been presented in the applied kinesiology literature.^{16, 32} To determine if an imbalance in electron poisoning is responsible, oxidating substances such as vitamin C or reducing factors like vitamin E are given to the patient to determine if the positive K27 therapy localization is abolished. Other endocrine factors are tested in the usual applied kinesiology manner, with K27 therapy localization added. Find the factor(s) which neutralize the positive K27 therapy localization.

Mental

The most common mental association with switching is the homolateral crawl pattern briefly mentioned on page 513 of this text and in Volume I. It is thoroughly discussed in Volume V. This is seen when a patient has a positive therapy localization with the right hand on the left K27 and the left hand on the right K27, termed a cross K27.

A positive K27 may develop as a result of a patient being under mental stress; therefore mental receptors have been added to the general classification of receptors in the body. When body language indicates mental stress, it can often be confirmed by having the patient therapy localize to K27 using one hand, with the thumb on one K27 and the index finger on the other. The other hand is used to therapy localize the two emotional neurovascular centers on the forehead. If this neutralizes the positive K27, there is evidence that there may be emotional stress.

nized by therapy localization to K27. Often a patient is tested for therapy localization to K27 and found negative in the clear. He may then be classified not switched, which is correct under those circumstances. A patient must be evaluated under the

Relation of the Stomatognathic System With Various Health Problems

conditions in which he lives. In other words, a patient who develops back pain throughout the day but obtains relief when he goes home and lies down will certainly not show the problems causing the back pain when he is supine; that is the position in which relief is obtained. Obviously the stress responsible for the health problem is not present in that position. That same individual might show negative therapy localization to K27 when supine, but positive when tested standing.

The use of K27 as a diagnostic tool helps to rapidly locate primary conditions. A patient is tested under various conditions of physical, chemical, or mental stress to find the cause of a positive K27. When the disturbance is located, standard diagnostic methods — palpation, laboratory, x-ray, and other factors of physical diagnosis — should be used to confirm the diagnosis before treatment is instituted.

As stated above, almost all — if not all — patients with chronic health problems which do not respond to treatment exhibit neurologic disorganization at some time and under certain circumstances. It is a physician's diagnostic acumen which enables him to find the neurologic disorganization and its cause by therapy localization to K27. When these procedures for finding hidden switching were first used, neurologic disorganization was found in only about 50% of the patients examined for it. As more and more factors causing neurologic disorganization were discovered and associated with body language, a higher percentage was found to be neurologically disorganized; the condition is currently found in almost 100% of the chronic, non-responding patients evaluated for it.

Examples are given here of the ways neurologic disorganization — structural, chemical, and mental — is found in some conditions. The illustrations show some of the major causes of neurologic disorganization; however, the final ability of a physician to locate it will be the result of the total study of applied kinesiology. As neurologic disorganization is more thoroughly understood, it appears that it is a classic example of Harper's²⁰ comment, "Anything can cause anything."

The examination begins with an individual who is negative to therapy localization at K27 in the clear. Various factors which may stress the body are added, and the patient is again tested for a positive K27. When the factor creating a positive K27 is located, it is more thoroughly evaluated to confirm its role in causing neurologic disorganization. Very often there will only be one factor creating a positive K27; however, in severe or chronic cases numerous factors may be discovered.

A physician's knowledge of body language signifi-



18—2. A standing test of the neck flexors may reveal weakness when K27 is therapy localized, although the muscles test strong without TL. Furthermore, they may test strong when supine with or without TL.

cantly reduces the time needed to locate the cause of switching. This is one of the reasons that hidden neurologic disorganization is found in a higher percentage of patients by doctors thoroughly knowledgeable in applied kinesiology procedures. Listening to the patient for clues to possible causes of neurologic disorganization is vital; it is possibly even more important to observe the patient as he describes his condition, moves about the room, and responds to the various stresses applied to his body.

Structural

It has already been mentioned that a patient may show a negative K27 when supine, but upon standing it becomes positive. Under these circumstances, test the patient in a seated position; this eliminates weight bearing in the feet and legs. If positive, there is the probability of pelvic or spinal involvement. Two-handed therapy localization can be used to quickly locate the problem area in most cases. While seated, the patient continues to therapy localize with one hand over one or both K27 points. Either or both can be positive; in some cases there is positive therapy localization only when both are touched simultaneously. The other hand is used to therapy localize different areas of the pelvis and spinal column to find

Relation of the Stomatognathic System With Various Health Problems

what negates the positive K27 therapy localization. The neck flexors or extensors or quadriceps can be used as indicator muscles. When the area is found, it is examined in the usual manner for subluxation or fixation, confirming the involvement with palpation and other procedures commonly used in chiropractic. If the subluxation or fixation is responsible for the switching, challenge will eliminate the positive TL to K27. If the final determination shows a subluxation or fixation in that area that is responsible for the switching, adjust the vertebra(e) as indicated by the challenge or fixation analysis. Test K27 again for positive therapy localization. If the adjustment was successful the patient will no longer test positive at K27 when seated.

If the patient exhibits negative K27 in the seated position, he is then tested while standing. Here the indicator muscles are limited to the neck flexors or extensors unless single-handed therapy localization to K27 is done, in which case an arm is freed. If positive, there is probable foot dysfunction, or possibly knee or pelvic involvement. There could be local disturbance in the muscles, usually of the neuromuscular spindle cell, but there could also be a fascial or trigger point problem. There are several ways to determine where the disturbance is and to specifically link it with the positive K27 in the standing position. The easiest way to examine this is with the patient supine. Again, K27 is therapy localized and should be negative. The articulations of the foot are challenged in various directions while the patient holds therapy localization on K27. Weakening of a previously strong indicator muscle is possible evidence of that challenge relating with neurologic disorganization. The challenge is repeated without therapy localization to K27; an indicator muscle may or may not weaken. If the indicator muscle no longer weakens without the therapy localization to K27, there is positive evidence that the challenge is to a joint subluxation which is creating neurologic disorganization. If the challenge still weakens the indicator muscle, further evaluation must be made to specifically tie the challenge with neurologic disorganization. This can be done by taping the foot or placing scaphoid, metatarsal, or lateral longitudinal pads under the foot to give support when standing. If, with this support, the patient no longer has a positive K27, evidence is that the foot dysfunction is responsible for neurologic disorganization.

An easier, but less specific, method of associating the foot with neurologic disorganization is to simply have the patient stand and rotate his leg so that the weight is carried on the lateral border of the foot, taking strain off the medial longitudinal arch and metatarsal heads. If foot pronation or dropped



18—3. Foot pronation which could be responsible for neurologic disorganization.



18—4. In some cases, putting weight onto the lateral longitudinal arches takes the strain out of the medial longitudinal and metatarsal arches and temporarily eliminates the positive TL to K27.

metatarsals are responsible for the neurologic disorganization, there will no longer be a positive therapy localization to K27 in the standing position. When there is evidence of foot or other subluxations, or muscle dysfunction in the legs responsible for neurologic disorganization, follow up with the usual diagnostic procedures and make corrections when indicated. The patient should no longer have a positive therapy localization to K27 following the therapeutic endeavor.

If there is positive evidence of neurologic disorganization in the sitting or standing position, but it does not appear to correlate with subjects previously discussed, consider a disturbance in the PRYT patterns. Often there can be modular stress in the body which does not cause a problem as long as there is no weight bearing. If body language indicates

Relation of the Stomatognathic System With Various Health Problems

this type of problem, have the patient lie supine and test him for the PRYT patterns, with and without therapy localization to K27. If a PRYT pattern is positive with therapy localization to K27 but not without, there is evidence that the positive pattern is contributing to neurologic disorganization. If it is positive both with and without K27 therapy localization, then two-handed therapy localization or challenge to the structure to be treated in that particular pattern will differentiate whether the positive pattern is associated with neurologic disorganization.

When there is no positive therapy localization to K27 in the standing position, yet there is body language of a problem with weight bearing or gait, the patient is placed into a simulated gait position and re-tested for positive K27. A more dynamic method of evaluating the gait mechanism for contribution to neurologic disorganization is to have the patient walk while therapy localizing K27. When he stops, test a strong indicator muscle — usually the neck flexors — for weakening. Involvement with the gait mechanism will more often be found when the patient walks in a figure eight pattern so that both right and left turning are included in the activity. Sometimes a problem will be missed if the patient simply walks in a straight line and does not turn in both directions. If a positive K27 develops with this type of stress, the patient should be evaluated for gait disturbances as previously discussed, similar to the method of evaluating for PRYT patterns. (For more in-depth discussion of weight bearing, ankle, foot, and gait problems, see Volume IV.)

Another area frequently involved with hidden neurologic disorganization is the stomatognathic system. Often a patient will show evidence of neurologic disorganization only while chewing, or perhaps very briefly during and immediately following deglutition. Generally a patient is tested to determine if the stomatognathic system is contributing to neurologic disorganization after the system has been thoroughly evaluated and treated. In this case, the various components of the system should test normal. They are now re-tested in the same manner previously discussed in this text, but in combination with therapy localization to K27. There will often be a positive finding under these circumstances. The positive factor is treated in the usual manner, and there should no longer be evidence of disturbance when tested in combination with K27 therapy localization.

In addition to the weight-bearing mechanism, gait, and the stomatognathic system, any other structural dysfunction within the body can contribute to neurologic disorganization; the contributing factors discussed here are problems most commonly found

on the structural side of the triangle.

Chemical

It is common for a patient's health problem to be caused by some chemical factor to which he is subjecting himself. This does not refer only to obnoxious chemicals, such as cleaning solution, paint, insecticides, alcohol, etc.; it also includes nutritional supplements. By taking massive doses of vitamin C, a patient can imbalance the electron poisoning system to the point that a chronic health problem develops. This may go unnoticed in the usual examination, but it is often found in the dynamic method of evaluating for neurologic disorganization. Again, the patient has been tested for positive therapy localization to K27 and found negative in the clear. If taking vitamin C causes a previously strong indicator muscle to weaken, there is presumptive evidence that the patient is over-oxidized and further vitamin C worsens the condition. Conversely, a weakening with vitamin E indicates the patient is over-reduced, and vitamin E worsens the condition. If there is no change in an indicator muscle after a patient tastes either complex, the test can be done in combination with therapy localizing K27. Sometimes one of the substances will cause positive therapy localization to K27, indicating that the patient is either over-reduced or over-oxidized; this contributes to neurologic disorganization. The patient should be evaluated thoroughly and procedures instituted to balance the electron poisoning system.¹⁶

In a similar manner, many different types of nutritional and chemical substances can be tested to determine if they adversely affect body function. This is a procedure used in applied kinesiology for many years. It can be combined with therapy localization to K27 to determine if there is adverse stimulation to the chemoreceptors of the body, thus creating neurologic disorganization.

Whenever nutritional or adverse chemicals are tested using applied kinesiology methods, the results should be correlated with standard physical diagnosis, which may include laboratory tests to confirm the findings. All factors of an examination should correlate before a final decision is made. Relying only on one test, such as a muscle weakening when a substance is chewed, is an inadequate procedure because variables might be present. For example, one may think that an individual weakens because of vitamin C when, in reality, there is weakness because of the carrier with which the vitamin C is mixed to form a tablet, or there may be an artificial sweetener in a chewable vitamin C to which the individual is sensitive. Use of these testing procedures is viable when combined with other diagnostic procedures in

which the physician should have expertise.

In some individuals who have body language suggesting endocrine disturbance, a positive K27 will develop when the lights are turned out, indicating pineal involvement. This weakening of an indicator muscle may not be present with the lights out if K27 is not therapy localized. The pineal gland¹⁶ is challenged and therapy administered, if necessary. The patient should then not weaken with the lights out when therapy localizing to K27. (The pineal gland is discussed in Volume V.)

There are many additional ways that nutritional and chemical factors can cause neurologic disorganization. The basic procedures for examining the various factors are similar to those presented here, and are discussed more thoroughly in Volume V.

Mental

As previously mentioned, the mental side of the

triad of health can show evidence of neurologic disorganization as observed by positive therapy localization to K27. There has not been as much clinical use of this aspect as of the chemical, and especially structural, factors. The primary use is with cross K27 therapy localization, which is discussed in Volume V and will not be expanded here. The primary clinical correlation of the mental side with therapy localization to K27 is that which is negated by two-handed therapy localization to K27 and the emotional neurovascular reflexes on the forehead.

The use of K27 as a diagnostic tool in finding the cause of neurologic disorganization often leads a physician to primary conditions very quickly. Observation of body language is the primary factor guiding the examination.

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Relation of the Stomatognathic System With Various Health Problems

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Index

— A —

Abducent n. (VI) (see also cranial nerves), 113
 Accessory bones (see wormian bones)
 Achlorhydria, 512
 Acoustic n. (VIII) (see also cranial nerves), 125-127
 Adenoid facies, 450
 Adrenal gland dysfunction influence on stomatognathic system, 239
 Alexander technique, 234
 Allergies, 512
 Adenoid facies, 450
 Calcium injections, 512
 Hypochlorhydria, 405
 Mouth breathing, 450
 Alpha condyle position, 308
 Alternate cover test, 114
 Alveolar bone remodeling in malocclusion, 347
 Anaglyph eye test, 115
 Angle classifications, 230-231
 Anosmia, 108
 Frontal lobe tumor, 108
 Trauma, 108
 Antacid medication relation to cranial faults, 176, 221
 Arteritis, temporal, 413
 Arthritis, TMJ, 328
 Gouty, TMJ, 410
 Infectious, TMJ, 411
 Osteoarthritis, TMJ, 328
 Psoriatic, TMJ, 411
 Rheumatoid, TMJ, 410
 Arthrography, TMJ, 329
 Articular eminence of TMJ, 53
 Articulating fossa (see mandibular fossa)
 Articulating surfaces in cranium, 76
 Asterion, 30
 Atlanto-occipital counterforce, 219
 Challenge, 219
 Correction, 219
 Sacral wobble correlation, 218, 219
 Therapy localization, 219
 Universal cranial fault correlation, 189
 Auditory
 Cochlear n., spiral organ of corti, 125
 Mandibular-malleolar ligament, 291
 Auditory disturbances, 512
 Body language of cranial faults, 140
 Body language of stomatognathic dysfunction, 479
 Cochlear n., 125
 Costen's syndrome, 232
 Etiology, 234
 Examination for, 126
 Mandibular-malleolar ligament, 300
 Narrowing of eustachian tube, 512
 Poor speech clarity, 512
 Rinne's test, 126
 Tensor palati and tympani m's, 412
 Tinnitus, 23, 126
 Weber's test, 126
 Auditory loss with learning disabilities, 513
 Auricular surface, sacroiliac, 200
 Axial loading of teeth, optimum occlusion, 344

— B —

Babinski-Weil test, 127
 "Banana head" cranial fault (see cranial faults, temporal bulge)
 Basic biological laws related to occlusion, 344
 Basion, 296

Bell's palsy, 122
 Recovery rate, 124
 Birth trauma, 24
 Cranial faults
 Malocclusion, 349
 Iatrogenic, 25
 Indicating possible cranial faults, 143
 Bite plane (bite plate or splint), 365-375
 Bruxism, 374
 Effects on cranial faults, 19
 Equilibration with cranial correction, 370
 Establishing physiologic vertical dimension, 370-374
 Fabrication, 369
 Increased vertical dimension,
 Temporary reduction in symptoms, 373
 Mandibular, illustration, 369
 TMJ disc dysfunction, 336
 Maxillary
 Illustration, 366
 Re-creating cranial faults, 368
 TMJ disc dysfunction, 336
 Maxillary arch, 19
 Neuromuscular imbalance therapy, 365
 Occlusal level adjusting, 373
 Protection of teeth, 374
 Reducing periodontal ligament stimulation, 365
 Remodeling of teeth without use of, 363-364
 Supplemental therapy
 Mechanical considerations, 367
 Neurologic considerations, 367
 Bite plate (see also bite planes), 365
 Bite, closed, 229
 Bite, open, 229
 Cheek biting, 452
 Lateral tongue thrust, 455
 Bite guards (see bite plane)
 Biting, incisor
 Masseter m., 247
 Muscles involved, 245
 Blood pressure
 Cranial dysfunction, 22, 172-173
 Blood sugar handling stress, periodontal disease, 239
 Blood vascular circulation as affected by malocclusion, 236
 Body language
 Bracing, 480
 Bruxism, 480
 Cranial faults (see also cranial faults, body language), 479
 Malocclusion, 480
 Neurologic disorganization, 481
 Orofacial m's imbalance (see muscles, mouth, body language)
 Stomatognathic area, 480
 Stomatognathic system dysfunction, 478-481
 TMJ dysfunction, 480
 Body-on-head reflexes, neurologic disorganization, 286
 Boley gauge, 255
 Bone flexibility, 12-15
 Cancellous bone, 13
 Bone torsion *in vivo*, 12
 Bottle-feeding
 As etiology of tongue thrust, 448
 Effect on maxillary arch, 450
 Flow rate of fluid, 448-450
 Importance of nipple design, 450
 Picard's mouth, 449
 Studies in children, 448
 Tongue action during, 449

Bracing, 352
 Body language of stomatognathic dysfunction, 480
 Etiology of referred pain to TMJ, 413
 Hypertonic masseter m., 408
 Iatrogenic considerations, 288
 Increasing vertical dimension, 371
 Masseter m., 248
 Mental/emotional stress, 240
 Temporalis m. hypertonicity, 246
 Tooth intrusion, 372
 Vertical dimension reduction, 254, 357
 Bradycardia
 Glossopharyngeal neuralgia, 129
 Vagus n. (X), 130
 Brain
 Circulation to, 22, 76
 Left vs. right activity, hyoid muscle involvement, 394
 Oxygenation of, 22
 Brain growth-spurt, 25
 Brain, bilateral activity, phonation, 435
 Branchial arches, 77-78
 Breast-feeding
 Adopted children, induced lactation, 448
 Advantages, 448
 As cranial motion enhancer, 25
 Buccinator m., 251
 La Leche League International, 448
 Natural vs. artificial nipple, 449
 Nipple protractility, 448
 Tongue action during, 449
 Breath holding, body language of cranial faults, 142
 Breathing, mouth
 Adenoid facies, 450
 Allergies, 450
 Detrimental habits, 451
 Effect on myofunctional therapy, 461
 Influence on stomatognathic system, 352
 Orbicularis oris m. weakness, 451
 Orofacial muscular imbalance, 450-451
 Sleeping habits, 451
 Tongue position, 451
 Breathing, nasal
 Difficulty, 182
 External frontal fault, 185
 Glabella cranial fault, 171
 Procerus m., 96
 Universal cranial fault, 187
 Breathing, oral, labella cranial fault, 171
 Bregma, 65
 Bruxism
 Artificially induced, 374
 Bite plane, protection of teeth, 374
 Body language of stomatognathic dysfunction, 480
 Definition, 357, 374
 Etiology, 374
 Etiology of periodontal disease, 348
 Etiology of referred pain to TMJ, 413
 Hypertonic masseter m., 408
 Iatrogenic considerations, 288
 Increased vertical dimension, 371
 Malocclusion, 374
 Masseter m., 248
 Masticatory muscle hypertonicity, 374
 Mobilization of cranial mechanism, 375
 Need for differential diagnosis, 352
 Poor lymphatic drainage, 375
 Psychophysiological, 374
 Vertical dimension reduction, 254, 357
 Buccal surface of tooth, 228

Index

— C —

- "Cadaveric position" of vocal cords, 132
- Calcium absorption, 512
- Calcium deficiency, hypochlorhydria, 405
- Cancellous bone, sphenobasilar junction, 13
- Carbohydrate, refined, periodontal disease, 348
- Carbon dioxide, influence on vasomotor regulation, 22
- Cardinal positions of gaze, 114
- Carotid canal, 55
 - Vagus n. (X), 130
- Carotid sinus, glossopharyngeal n. (IX), 128
- Carotid sinus stimulation
 - Glossopharyngeal n. (IX), 128
 - Vagus n. (X) dysfunction, 132
- Category pelvic faults (see pelvic faults)
- Cellulome therapy, 418
- Centric occlusion (see also occlusion)
 - Anterior slide, normal, 308
 - Definition, 229
- Centric relation
 - Definition, 229
 - Optimum occlusion, 344-345
 - Physiologic, 308, 376
- Cerebrospinal fluid, 75
 - Blood pressure, 22
 - Blood vascular function, influence on, 76
 - Cranial faults influence on, 20
 - Flow, 76
 - Fluctuation in cranial motion, 150
 - Meninges effects on, 75
 - Pituitary gland secretions, 20
 - Production of, 75
 - Secondary circulatory system hypothesis, 20
 - Secretion of, 76
 - Spinal nerves, flow along, 20
- Cervical traction
 - Effect on TMJ, 411
 - Iatrogenic conditions, 287
- Challenge
 - Cranial faults (see specific cranial fault)
 - Cranial faults, general, 153-154
 - Hyoid muscles, 395-399
 - Sutures of eye socket, 94
- Cheek biting, 452
- "Cheerleader's Syndrome," 410
- Chemical imbalances
 - Etiology of cranial faults, 27
 - Relation to cranial faults, 221
 - Relation to stomatognathic system, 239
- Chemoreceptors, 283
- Chewing
 - In diagnosing TMJ dysfunction, 435, 437
 - Soft vs. hard foods, 247, 437
- Children, incidence of tongue thrust, 447
- Circulation, blood vascular, effects of malocclusion, 236
- Cloacal synchronization
 - Cranial influence, 170
 - Cranial-sacral correlation, 214-215
- Closed kinematic chain
 - Block diagram of muscles, 281
 - Example of muscle imbalance, 354
 - Head-to-shoulder girdle muscles, 275-282
 - Muscles of, 280
 - Occlusal and muscle imbalance, 235
- Closest speaking space, 304, 373
- CO (see centric occlusion)
- Coccygodynia, 210
- Coccyx (see also pelvic faults, coccyx)
 - Anatomy, 199
 - Lovett brother, 203
 - Motion, 203
- Condyle, mandible (see mandible, condyle)
- Condylectomy, effects on mandibular closing engram, 351
- Condylusquomastoid pivot, 53, 57
- Confrontation test, 110
- Conjunctivitis, 512
- "Core link," 202
- Corneal reflex test, ophthalmic n., 120
- Costen's syndrome, 232
- Cover test, 114
- CR (see centric relation)
- Cranial anatomy (see cranial osteology, nerves, etc.)
- Cranial bones (see individual bones)
- Cranial fault etiology, 24-27
 - Birth trauma, 24, 52
 - Chemical imbalances, 27
 - Cranial muscles, 27
 - Dental procedures, 26
 - Habit patterns, 27
 - Head injuries, 25
 - Iatrogenic considerations, 26
 - Malocclusion, 311
 - Myofascial system, 27
 - Remote, 148
 - Structural imbalance, 26
 - Whiplash, 25
- Cranial faults
 - Anosmia, 108
 - Auditory disturbances, 23
 - Auto-correction, 271
 - Bell's palsy, 142
 - Bite plane therapy considerations, 367-370
 - Maxillary vs. mandibular bite planes, 368
 - Blood pressure disturbances, 172
 - Body language, 480
 - Auditory disturbances, 142-148
 - Breath holding hiding indications of, 142
 - Bruxism, 144
 - Ear, external flaring, 146
 - Eye protrusion, 146
 - Headaches, 143
 - Mandibular position, 147
 - Nares flaring, 146
 - Nasolabial fold, 147
 - Neurologic disorganization, 142
 - Pain along sutures, 147
 - Patient consultation, 143-144
 - Posterior observations, 147
 - Remote problems, 148
 - Skull asymmetry, 144
 - Skull symmetry, 144-147
 - Temporal squama flaring, 146
 - Vertigo, 142
 - Visual disturbances, 142
 - "Whiplash"-type injuries, 143
- Cerebrospinal fluid, 20-22
- Cervical traction as etiology, 287
- Challenge
 - Rebound and static, 153-154
 - Reciprocal tension membrane, 154
- Challenge, general, 151, 153-154
- Cloacal synchronization correlation, 214-216
- Combined inspiration and expiration, 170-171
- Concussive manipulation, contraindications, 156
- Correction evaluated by dental procedures, 18
- Correction prior to equilibration, 377
- Correction, general, 155-157
- Corrective effort evaluation, 156
- Cranial nerve dysfunction, 20
- Dental splints, 19
- Cranial faults (continued)
 - Dominant over occlusion, 349-350
 - Endocrine disturbances, 22
 - Equilibrium reflexes, 23
 - Error in occlusion examination, 423-424
 - Examination and treatment, 139-195
 - Examination methods, 148-151
 - Applied kinesiology, 150
 - Osteopathic, 149
 - Palpation, 149
 - Visual observation, 148
 - Expiration assist, 162-164
 - Challenge, 163
 - Correction, 164
 - Mechanics of correction, 162
 - Mechanics of fault, 162
 - Muscle correlation, 164
 - Pain location, 164
 - Sphenoid and occipital bone movement, 140
 - Therapy localization, 163
 - Expiration, forced, 167
 - External frontal, 184-186
 - Challenge, 185
 - Conditions correlating, 185
 - Correction, 186
 - Mechanics of correction, 184
 - Mechanics of fault, 184
 - Muscle correlation, 185
 - Pain location, 185
 - Rhinitis, 185
 - Therapy localization, 185
 - Eye protrusion, 180
 - Eye retrusion, 180
 - Flexion and extension faults, 157-173
 - Glabella, 171-173
 - Challenge, 172
 - Conditions correlating, 172
 - Correction, 173
 - Mechanics of correction, 171
 - Mechanics of fault, 171
 - Muscle correlation, 172
 - Pain location, 172
 - Sacral fault correlation, 173
 - Therapy localization, 171
 - Gum chewing hiding indications, 375
 - Hidden, 490
 - Hypertension, 22, 172
 - Glossopharyngeal n. (IX), 129
 - Iatrogenic condition prevention, 155-156
 - Iatrogenic considerations, 148, 156, 287-289
 - Dental procedures, 23, 144
 - Idiopathic scoliosis, 23
 - Inspiration assist, 157-161
 - Challenge, 159
 - Correction, 160
 - Mechanics of correction, 158
 - Mechanics of fault, 158
 - Muscle correlation, 159
 - Pain location, 160
 - Sphenoid and occipital bone movement, 140
 - Therapy localization, 159
 - Inspiration, forced, 165
 - Interaction with occlusion, 368
 - Internal frontal, 180-184
 - Challenge, 181
 - Conditions correlating, 182
 - Correction, 183
 - Definition, 180
 - Mechanics of correction, 180
 - Mechanics of fault, 180
 - Metopic suture, 180
 - Muscle correlation, 182

- Cranial faults (continued)
 Pain location, 182
 Retruded eye, 180
 Rhinitis, 182
 Therapy localization, 181
 Interosseous, 52, 147
 Introduction, 139
 Lambdoidal suture, 193
 Challenge, 193
 Conditions correlating, 193
 Correction, 193
 Muscle correlation, 193
 Pain location, 193
 Therapy localization, 193
 Lasting correction, 157
 Mental/emotional stress, 240, 288
 Nasal respiration, 171
 Neurologic disorganization, 23
 Neurologic disorganization etiology, 285
 Occlusion, effects on, 270, 348-350
 Optimum first approach, 157
 Oral respiration, 171
 Osteopathic approach, 149
 Pain patterns, general, 155
 Palpation for lesions, 149
 Parietal descent, 177-179
 Challenge, 178
 Conditions correlating, 179
 Correction, 179
 Finding hidden faults, 178
 Half-breath held out, 177
 Headache, temporal, 178
 Mechanics of correction, 178
 Mechanics of fault, 177
 Muscle correlation, 178
 Pain location, 178
 Sagittal suture fault, iatrogenic, 179
 Temporal bulge correlation, 179
 Therapy localization, 178
 Patient consultation, methodology, 143-144
 Pelvic correlation, 197
 Perpetuation by remote problems, 148
 Pineal gland, effects on, 22
 Pituitary gland, effects on, 22
 Recidivism, 5, 153, 220-222, 271-272
 Neurologic tooth, 266
 Reciprocal tension membrane, 139
 Reproducible corrections, 157
 Respiratory correlation, 139-141
 Role in health and dysfunction of, 20-24
 Rotational faults, 174-189
 Sagittal suture, 190-191
 Challenge, 190-191
 Conditions correlating, 191
 Correction, 191
 Mechanics of correction, 190
 Mechanics of fault, 190
 Muscle correlation, 190-191
 Pain location, 190
 Therapy localization, 190-191
 Sensation felt by patient with correction, 161
 Sphenobasilar expiration assist, 167-169
 Challenge, 168
 Correction, 169
 Finding hidden faults, 169
 Mechanics of correction, 167
 Mechanics of fault, 167
 Muscle correlation, 169
 Pain location, 169
 Therapy localization, 168
 Sphenobasilar extension fault (see sphenobasilar inspiration assist)
 Sphenobasilar flexion fault (see sphenobasilar expiration assist)
- Cranial faults (continued)
 basilar expiration assist)
 Sphenobasilar inspiration assist, 165-167
 Challenge, 166
 Correction, 167
 Finding hidden faults, 166
 Mechanics of correction, 165
 Mechanics of fault, 165
 Muscle correlation, 166
 Pain location, 167
 Therapy localization, 166
 Squamosal suture, 191-192
 Challenge, 191
 Conditions correlating, 192
 Correction, 192
 Muscle correlation, 191
 Pain location, 192
 Temporalis m. hypertonicity, 246
 Therapy localization, 191
 Stomatognathic sequential correction, 487
 Stomatognathic system, 144
 Sutural faults, 190-195
 Swallowing eliminating evidence of, 271
 Temporal bulge, 146, 174-176
 Antacid tablets, 176
 "Banana head," 174
 Challenge, 175
 Conditions correlating, 176
 Correction, 176
 Ear, flaring of, 175
 Finding hidden faults, 175
 Half-breath held in, 174
 Masking with hydrochloric acid tablets, 176
 Mechanics of correction, 175
 Mechanics of fault, 174
 Muscle correlation, 175
 Parietal descent correlation, 176
 Respiratory correlation, 174
 Therapy localization, 175
 Vagus n. (X) involvement, 176
 Therapy localization
 General, 151, 152-153
 Limitations of, 487
 Thoracic outlet syndrome, 178
 Thoracic respiratory correlation, 35
 Three-dimensional characteristics, 154, 159, 162
 Tooth extraction, 287
 Universal, 187-189
 Challenge, 187
 Conditions correlating, 188
 Correction, 189
 Mechanics of correction, 187
 Mechanics of fault, 187
 Muscle correlation, 188
 Pain location, 188
 Therapy localization, 187
 Vector of correction, 154, 156
 Visual disturbances, 23
 "Whiplash," 113
 Zygomatic suture, 194-195
 Challenge, 194
 Conditions correlating, 194
 Correction, 194-195
 Muscle correlation, 194
 Pain location, 194
 Therapy localization, 194
- Cranial motion, 35-38
 Autonomous cycling, 18
 Birth trauma, 39
 Bone flexibility, 13
 Cardiac pulse correlation, 17
- Cranial motion (continued)
 Cerebrospinal fluid, 20
 Cerebrospinal fluid fluctuation, 150
 Condylusquamosal (CSM) pivot, 53, 57
 Cranial bone movement, correlation of, 79-83
 Cranial rhythmical impulse, 149
 Cranial vault, 79-82
 Cyclic, 9
 Electrical oscillations, 19
 Embryology, 77-78
 Equilibration, effects of, 18
 Ethmoid bone, 70, 81
 Axis of rotation, 81, 85
 Evaluating normal motion, 87
 Evaluation of, 87-88
 Expiration, 35, 79
 Extension, 79
 Flexion, 79
 Foramen magnum, 79
 Frontal bone, 38, 61, 83
 Axis of rotation, 61, 83
 Coronal suture, 83
 External rotation, 83
 Glabella, 83
 Internal rotation, 83
 Metopic suture, 83
 History of discovery, 9
In vivo bone torsion, 13
 Individuality, 83, 87-88
 Inspiration, 35, 79
 Integration of, 76-78
 Introduction, 10
 Involuntary active motion, 149
 Mandible, 87
 Mastication aiding in, 271
 Maxillary arch measurement, 18
 Maxillary bone, 67, 84
 Dental arch, 84
 Ethmoid bone, 84
 Intermaxillary suture, 84
 Lacrimal bone, 84
 Nasal bone, 84
 Vomer bone, 84
 Measurement in monkeys, 18
 Meninges, effects of, 75
 Movement between sutures, 13
 Nasal concha, 73
 Objective measurement, 17-20
 Objective measurement, first, 17
 Occipital bone, 35, 37, 52
 Axis of rotation, 79
 Hindering motion, 87
 Squama, 79
 Osteopathic approach, evaluation of examiner, 150
 Osteopathic diagnostic parameters, 19
 Palatine bone, 69, 86
 Interpalatine suture, 86
 Pyramidal process, 86
 Palpation for, 10
 Parietal bone, 65, 83
 Lambdoidal suture, 83
 Parietomastoid suture, 83
 Squamosal suture, 83
 Passive motion, 149
 Pelvic activity, 9
 "Physiologic motion," 149
 Predictability, 10
 Psychiatric patients, study of, 513
 Respiration parallel, 9
 Respiratory correlation, 139-141
 Restricting normal motion, experimental, 140
 "Rock shaft and pivot," 57, 79

Index

- Cranial motion (continued)
 - Rotation, 79
 - Sacral motion correlation, 18
 - Self palpation, 16
 - Sphenobasilar extension, 35, 79
 - Sphenobasilar flexion, 10, 35, 37, 79
 - Sphenobasilar junction, 10, 37, 79
 - Sphenoid bone, 37, 48
 - Axis of rotation, 35, 79
 - Key bone in motion, 43
 - Sphenosquamous pivot, 35, 48, 49, 57, 59, 79, 81
 - Spinal motion, effects on, 18
 - Sutural development, 88
 - Sutural nerve receptors, 20
 - Suture fixation experiment, 15
 - Suture flexibility, 42
 - Temporal bone, 10, 36, 37, 56, 57, 79
 - Axis of rotation, 56, 79, 80
 - Hindering motion, 87
 - Mastoid portion, 80
 - Mastoid process, 80
 - Occipitomastoid suture, 80
 - Petrous portion, 79
 - Squama, 80
 - "Wobbling wheel effect," 80
 - Thoracic respiratory correlation, 17
 - "Unphysiologic motion," 150
 - Vomer bone, 71, 85
 - "Wobbling wheel effect," 56
 - Wormian bones, 25, 57
 - X-ray evaluation, 18
 - Zygomatic bone, 70, 87
- Cranial nerves, 105-138
 - Abducent n. (VI)
 - Etiological case studies, 113
 - Examination, 114-115
 - Acoustic n. (VIII), 125-127
 - Cochlear n., 125
 - Examination, 126-127
 - Possible entrapment, 126
 - Vertigo, 512
 - Vestibular n., 125
 - Ansa cervicalis n., 135
 - Artificial synapse, 107
 - Body language of stomatognathic dysfunction, 479
 - Chorda tympani n., 122
 - Cochlear n.
 - Evaluation of, 126
 - Rinne's test, 126
 - Weber's test, 126
 - Compression block, 20
 - Entrapment, pathological vs. functional, 105
 - Examination of III, IV, and VI, 113-114
 - Facial n. (VII), 122-124
 - Bell's palsy, 122-124
 - Cervical subluxation interference, 124
 - Chorda tympani n., 122
 - Examination, 122-124
 - Motor distribution, 122
 - Possible entrapment, 122
 - Sensory distribution, 122
 - General, 105-107
 - Glossopharyngeal n. (IX), 128-129
 - Carotid sinus, 128-129
 - Examination, 128
 - Gag reflex test, 128
 - Idiopathic hypertension, 76
 - Neuralgia, 129
 - Possible entrapment, 128
 - Hypoglossal n. (XII), 135-136
 - Ansa cervicalis n., 135
- Cranial nerves (continued)
 - Examination, 136
 - Possible entrapment, 136
 - Hypothesis of entrapment, 75, 107
 - Intercellular space, 107
 - Intrapontine lesion, 124
 - Introduction, 105
 - Mandibular n., 118
 - Maxillary n., 116-118
 - Cranial branch, 118
 - Facial branches, 118
 - Infraorbital canal branches, 118
 - Pterygopalatine fossa branches, 118
 - Nervus intermedius n., 122
 - Oculomotor n. (III), 111
 - Examination, 114-115
 - Possible entrapment, 111
 - Etiological case studies, 111
 - Olfactory n. (I), 70, 108
 - Examination, 108
 - Possible entrapment, 108
 - Ophthalmic n.
 - Frontal n. branch, 116
 - Lacrimal n. branch, 116
 - Nasociliary n. branch, 116
 - Optic n. (II), 109-110
 - Examination, 110
 - Possible entrapment, 110
 - Visual disturbances, 23
 - Otic ganglion, 122
 - Pathological vs. functional disorders, 136-137
 - Spinal accessory n. (XI), 132-134
 - Anatomy, 132
 - Body organization, 483
 - Examination, 134
 - Possible entrapment, 134
 - Spinal part, 132
 - Trigeminal n. (V), 116-121
 - Etiological case studies, 119-121
 - Examination, 120-121
 - Possible entrapment, 119-120
 - Semilunar ganglion, 116
 - Trochlear n. (IV), 112
 - Etiological case studies, 112
 - Examination, 114-115
 - Possible entrapment, 112
 - Vagus n. (X), 130-132
 - Dysphagia, 132
 - Examination, 130-132
 - Possible entrapment, 130
 - Temporal bulge cranial fault, 176
 - Vagotomy, 132
 - Vestibular n., 127
 - Vestibulocochlear n. (see acoustic n.)
 - Vidian n., 122
 - Visual disturbances, 114-115
- Cranial osteology, 29-88
 - Articular eminence of TMJ, 54
 - Asterion, 30
 - Basion, 296
 - Bone development, 39
 - Bone flexibility, 10
 - Bregma, 30
 - Cruciate suture, 33
 - Ethmoid bone, 70
 - External occipital protuberance, 31
 - Foramen lacerum, 54-55
 - Frankfurt plane, 482
 - Frontal bone, 61-63
 - General landmarks, 30-34
 - Anterior skull, 32
 - Inferior skull, 33
 - Internal cranial floor, 34
- Cranial osteology (continued)
 - Lateral skull, 30
 - Posterior skull, 31
 - Inion, 31
 - Jugular foramen, 54
 - Lacrimal bone, 73
 - Lambda, 31
 - Mandibular fossa, 54
 - Mastoid angle of parietal bone, 30
 - Mastoid notch, 31
 - Mastoid portion, 30, 54
 - Mastoid process, 30
 - Maxillary bone, 65-67
 - Metopic suture, 31
 - Metopic suture remnant, 32
 - Nasal bone, 72
 - Nasal concha, 72-73
 - Nuchal lines, 31
 - Occipital bone, 50-53
 - Occipital crest, 31
 - Orbitale, 482
 - Palatine bone, 67-69
 - Parietal bone, 64-65
 - Petrous portion, 54
 - Porion, 482
 - Pterion, 30
 - Sphenoid bone, 43-49
 - Temporal bone, 53-60
 - Temporal lines, 30
 - Tympanic portion, 55
 - Vomer bone, 71
 - Zygomatic bone, 69-70
- Cranial primary respiration, 35-38
 - Breathing correlation, 139-141
 - Bruxism as mobilizing factor, 375
 - Definition, 35
 - Expiration, definition, 35
 - Iatrogenic considerations, 286-289
 - Inspiration, definition, 35
 - Mandibular influence on, 270-273
 - Sphenobasilar extension, definition, 35
 - Sphenobasilar flexion, definition, 35
 - Stomatognathic area interdependence, 269-289
- Cranial rhythmical impulse, 149
- Cranial suture, histology of, 13
- Cranial syndesmology, 39-42
- Cranial therapeutics
 - Iatrogenic problems, 10
 - Names of approaches, 10
 - Practitioners of, 9
 - Symmetry of skull, 10
- Cranial vault, 79-82
- Cranial-pelvic movement correlation, 197
- Cranial-sacral primary respiration
 - Introduction, 3-7
 - Motion correlation, 202
 - Recurrent, 220-222
- Craniomandibular joint (see also temporomandibular joint), 294
- Cranium, muscles and fascia of, 89, 90-92
- Cribriform plate, 70
- Crista galli, 70
- Cross K27, 519
- Crossbite, mouth breathing etiology, 451
- Crossbite occlusion, 229
- Cruciate suture, 33
- CSM pivot (see condylosquamosomastoid pivot)
- Cyber II muscle testing, 236

— D —

Definitions, orthodontic nomenclature, 230-231

- Deflective tooth contacts (see also malocclusion)
 Anterior-superior, 344-345
 Lateral, 344
- Degenerative joint disease (see osteoarthritis)
- DeJarnette block technique, 211
- Dental arch
 Effects of bottle-feeding, 450
 Effects of mouth breathing, 451
 Homuncular representation, 263-265
 Muscular envelope, 251
 Orofacial muscular envelope, 442
- Dental chair headrest, 19
 Effects on equilibration, 359
- Dental distress syndrome, 6
- Dental procedures, iatrogenic cranial faults, 26, 287
- Dental terminology, 226-231
- Dentures
 Effect on cranium, 19
 Periodontal ligament, 260
 "Settling in," 357
 Tongue thrust, 446
 Vertical dimension determination, 372
- Diagnosis, conflicting information, 409
- Diaphoresis, gustatory sweating, vagus n. (X), 132
- Diaphragma sellae, 75
- Diaphragmatic respiration, definition, 35
- Diastemata, tongue thrust, 455
- Digestive disturbance, 512
 Antacid tablets, 176
 Body language of cranial faults, 140
 Hypochlorhydria, temporal bulge correlation, 176
 In infant, 143
 Lambdoidal suture cranial fault, 193
 Temporal bulge correlation, 176
 Toxic vascular headaches, 514
 Vagus n. (X) dysfunction, 132
- Digit sucking, habit pattern correction, 458
- Diplopia, 479
 Examination for, 115
- Disc dysfunction, TMJ, 328-339
 Arthrography analysis, 329
 Conservative treatment, 333-337
 Cranial fault influence on, 333
 Disc dislocation, 333, 425
 Disc displacement, 333, 425
 Examination methods, 329
 External pterygoid m., 334
 Indication by therapy localization, 425
 Malocclusion, 334
 Manipulation, 334-336
 Maxillary bite plane, 336
 Muscle balancing of system, 333
 Muscle imbalance, 329
 Nutritional support, 336
 Occlusion, 329
 Opening click, 331
 Opening lock, 332
 Recidivism, 336
 Reciprocal click, 331
 Surgical intervention, 337
 Temporalis m., posterior fibers, 333-334
 Trismus, 337
 X-ray analysis, 329
- Dislocation, TMJ, 411
- Disocclusion, optimum occlusion, 344
- Distal surface of tooth, 228
- Dizziness (see vertigo)
- "Doll eyes," 500
- Dura mater
 (continued)
 Anatomy, 197
 Influence on cranial-sacral motion, 201
- Dyslexia, 514
- Dysphagia, 130
 Vagus n. (X), 132
- E —
- Ear, etiology of referred pain, 412
- Edentulous patients, mandibular closing engram, 351
- EID (see eyes into distortion)
- Electromagnetic field, hyoid m. balance, 395
- Electromagnetic receptors, 283
- Electron poisoning in neurologic disorganization, 519, 522
- Embryology, cranial, 77-78
 Branchial arches, 77-78
 Cranial motion, correlation with, 77
- Emotional problems (see mental/emotional stress)
- Endocrine disturbances, 514
 Cranial motion dysfunction, 22-23
 Cranial nerve entrapment, 23
 Pineal gland, 523
 Pituitary gland, 514
 Two-hand therapy localization, 515
- Engram, mandibular closing
 Condylectomy, 351
 Edentulous patients, 351
 Influence on jaw closing, 503
 Masticatory muscles, 364
 Occlusion, 355
 Periodontal ligament proprioceptors, 351
- Envelope of motion, mandible (see mandibular motion)
- Envelope of teeth, 442
- EOD (see eyes out of distortion)
- Epicanthus, 114
- Epicranial aponeurosis, 91-92
- Epineurium, 74
- Equilibration, 375-378
 Basic procedures prior to, 377-378
 Bite plane therapy prerequisite, 367
 Closing engram dysfunction, 378
 Definition, 375
 Dental chair headrest interference, 359
 Effects on cranial motion, 18
 Examination procedures prior to, 359-375
 Masticatory muscles
 Balance system first, 377
 Electrical stimulation for relaxation, 378
 Patient's position during, 377
 Physiologic centric relation, 376
 Registering prematurities, 377
 Equilibrium, vestibular n., 125
- Equilibrium disturbances, 512
 Babinski-Weil test, 127
 Body language of stomatognathic dysfunction, 480
 Labyrinthitis, 127
- Equilibrium proprioceptors (see proprioceptors, equilibrium)
- Equilibrium theory of tooth position, 442-444
- Equilibrium, body on head, infrahyoid muscles, 390
- Esotropia, differential diagnosis, 114
- Ethmoid bone
 Articulations, 71
 General landmarks, 70
 Motion, 71, 84-85
 Olfactory n. (I), 70
- Ethology, 234
- Ethyl chloride, trigger point therapy, 417
- Eustachian tube blockage, auditory loss, 6000
 cycle level, 512
- Expiration, definition, 35
- Expiration assist cranial fault (see also cranial faults), 162
- Extension (see sphenobasilar extension)
- External frontal cranial fault (see also cranial faults, external frontal), 184-186
- Eye
 Pain behind, 182, 185
 Protruded
 Body language, 146-147
 External frontal fault, 184
 Retruded, internal frontal fault, 180
- Eyes into distortion (EID), 500-502
 "Doll eyes," 500
 Effect on body balance, 500
 Eyes out of distortion (EOD), 501
 Hyoid bone, 501
 vs. ocular lock, 501
- Eyes out of distortion (EOD), 501
- F —
- Face, muscles and fascia of, 89
- Facial grimacing, deviant swallowing pattern, 454
- Facial n. (VII), 122-124
- Facial surface of tooth, 228
- Falx cerebelli, 74
- Falx cerebri, 70, 74
 Frontal bone attachment, 61
- Fascia
 Continuity throughout body, 75, 89, 90
 Cranium, 90
 Cranium and face, 89
- Fascial release technique
 General description, 355
 Hyoid muscles, 396
 Hypertonic TMJ muscles, examination, 424
 Masticatory muscles, 420
 Nutritional supplementation, 420
- Fibrositis, referred pain to TMJ, 413
- Filum terminale, 198
 Attachment to coccyx, 203
- Fingernail biting, 451
 Habit pattern correction, 460
 Nutritional deficiency, 460
- Flexion (see sphenobasilar flexion)
- Fluori-Methane, trigger point therapy, 417
- Folic acid, periodontal disease treatment, 348
- Fontanelle
 Mastoid, 30
 Posterior, 31
- Foot dysfunction
 Etiology of neurologic disorganization, 516, 521
 Stomatognathic system dysfunction, 354
- Foramen lacerum, 34
- Force scale, orbicularis oris m. test, 452
- Force transducer, Myoscanner, 452
- Form vs. function
 Centric relation, 310
 Equilibration, 310
 Myofunctional therapy, 442
- Frankfurt plane, 482
- Freeway space
 Definition, 229
 Determination of, 305
 Factors which influence, 305

Index

- Freeway space (continued)
 - Importance of maintaining, 305, 371
 - Physiologic rest position, 305
 - Frenulum linguae, 256
 - Frontal bone
 - Articulations, 61-63
 - Axis of rotation, 61
 - General landmarks, 61-63
 - Glabella, 61-62
 - Metopic suture, 61, 62, 83
 - Motion, 38, 61, 83
 - Ossification centers, 61
 - Frowning, 95
 - Function vs. form (see form vs. function)
- G —
- Gag reflex test, 128
 - Gait exercises, 509
 - Gait mechanism, 505-509
 - Etiology of neurologic disorganization, 522
 - Gait exercises, 509
 - Reciprocal inhibition, 505
 - Weight-bearing test against hi-lo, 508
 - Galea aponeurotica, 91-92
 - Ganglion, nodose, 130
 - Ganglion, otic, 122
 - Ganglion, semilunar, 116
 - Entrapment, 119
 - Pathology of, 119
 - Garliner, Daniel, 441
 - Gastroenteritis, allergic, 512
 - Glabella, 61
 - Glabella cranial fault (see also cranial faults, glabella), 171
 - Glaucoma, cranial fault indications, 142
 - Glenoid fossa (see mandibular fossa)
 - Glossodynia, 136
 - Glossopharyngeal n. (IX), 128-129
 - Hypertension, idiopathic, 76
 - Neuralgia, 129
 - Glossopharyngeal neuralgia, 129
 - Guide planes (see bite planes)
 - Gum chewing
 - Hiding cranial faults, 375
 - Relation to cranial faults, 221
 - Gustatory sweating, vagus n. (X), 132
 - Gyroscope, hyoid orientation, 278
- H —
- Habit correction (see specific habit: thumb-sucking, lip-licking, etc.)
 - Habit patterns
 - Etiology of cranial faults, 27, 221
 - Etiology of malocclusion, 346
 - Orofacial muscle imbalance, 451
 - Periodontal disease, 348
 - Head injuries, cranial faults, 25
 - Head leveling
 - Closed kinematic chain, 280
 - Examination, 482
 - Postural muscles, 275-277
 - Spinal accessory n. (XI), 483
 - Head-on-shoulder girdle muscle interrelationship, 280-282
 - Head-to-shoulder girdle muscles, closed kinematic chain, 275-282
 - Headache, 514
 - Behind the eye, 182
 - Body language of cranial faults, 140, 142
- I —
- Iatrogenic conditions
 - Cervical traction, 287, 515
 - Prevention, 151, 358-359
 - TMJ, 412
 - Idiopathic scoliosis, neurologic disorganization, 23
- J —
- Jaw opening
 - Digastric m., 386
 - "Three-knuckle rule," 406
 - Jaw opening, maximal
 - Digastric m., anterior belly, 394
 - EMG studies of, 255
 - Jugular foramen, 54
- K —
- K27 switching (see neurologic disorganization)
- L —
- La Leche League International, 448
 - Labyrinthine reflexes, neurologic disorganization, 286
 - Labyrinthitis, 127
 - Lacerum foramen, 54
 - Lacrimal bone, 73
 - Lacrimal gland, 94, 122
 - Examination, 124
 - Sensory innervation, 116
 - Lacrimal sac, 73, 94
 - Innervation, 116
 - Lambda, 30, 65
 - Lambdoidal suture cranial fault (see also cranial faults, lambdoidal suture), 193
 - Larynx
 - "Cadaveric position" of vocal cords, 132
 - Movement, 390, 392
 - Vagus n. (X), 132
 - Learning disabilities, 513
- M —
- Ileocecal valve syndrome, toxic vascular headache, 514
 - Ileocecal valve, closed
 - Lambdoidal suture cranial fault, 193
 - Universal cranial fault, 189
 - Ileocecal valve, open, zygomatic suture cranial fault, 194
 - Ilium (see innominate bone)
 - Incisive papilla, 465
 - Infant health problems, 25
 - Inion, 31
 - Innominate bone (see also pelvis)
 - Articulation with sacrum, 200
 - Ilium, 199
 - Ischium, 199
 - Motion, 204
 - Motion from abdominal m's, 204
 - Pubis, 199
 - Inspiration, definition, 35
 - Inspiration assist cranial fault (see also cranial faults, inspiration assist), 157
 - Interincisal distance, diagnostic aid in TMJ dysfunction, 255
 - Internal acoustic meatus, 55
 - Internal frontal cranial fault (see also cranial faults, internal frontal), 180-184
 - Interocclusal distance (see also freeway space), 229
 - Interosseous cranial fault, 52
 - Ionization, 182
 - Iron, chelated, slow muscle fibers in TMJ movement, 423
 - Ischium (see innominate bone)

- Leg length
 - Atlas subluxation, 211
 - Category I pelvic fault, 211
 - Leg short, effects on occlusion, 235
 - Ligament interlink technique, hyoid m's, 394
 - Ligament, periodontal (see periodontal ligament)
 - Ligaments
 - Mandibular-malleolar, 291, 512
 - Sacroiliac, 200
 - Sphenomandibular, 299
 - Stylohyoid, 384
 - Stylomandibular, 299
 - Temporomandibular, 299
 - Thyrohyoid, 384
 - TMJ, 299-300
 - Lingual frenulum, 456
 - Lingual surface of tooth, 228
 - Lips
 - Chapping, 455
 - Licking, 460
 - Characteristics, 454
 - Open mouth posture, 454
 - Lisping, deviant swallowing pattern, 456
 - Logan basic technique, modified in PRYT technique, 498
 - Long centric, 318
 - Lovett brother
 - Occiput-sacrum, 203
 - Sphenoid-coccyx, 203-210
 - Luxation, TMJ (see also TMJ dislocation), 411
 - Lymphatic congestion
 - Etiology of bruxism, 375
 - Small intestine, 95
- M —
- Macroglossia, 456
 - Magnet, influence on hyoid m. balance, 395
 - Malnutrition
 - Etiology of malocclusion, 345
 - Mandibular growth, 356
 - Malocclusion
 - Anterior tongue thrust, 446
 - As etiology of tongue thrust, 452
 - Bite plane effect on body balance, 235
 - Bite plane therapy
 - Mechanical considerations, 367
 - Neurologic considerations, 367
 - Bite plane-plate-splint therapy, 365-375
 - Body balance, effects on, 235
 - Body language, 480
 - Bruxism, 374
 - Cranial fault etiology, 348-350
 - Cranial influence in examination, 423
 - Definition, 229
 - Dominant over cranial faults, 349-350
 - Effects on blood vascular flow, 236
 - Etiology, 345
 - Dental procedures, 358
 - Habit pattern, 346, 451
 - Iatrogenic, 358
 - Malnutrition, 345
 - Masticatory muscles, 352
 - Periodontal disease, 348
 - Referred pain to TMJ, 412
 - Remote factors, 233-234, 358
 - Vertebral manipulation, 358
 - Forced centric occlusion, 422
 - From AK techniques, 364
 - Incidence in children, 346
 - Malocclusion (continued)
 - Increased vertical dimension, 357
 - Intrusion of teeth, 357
 - Lip characteristics, 454
 - Loss of posterior support, 300
 - Mandibular Kinesiograph measurement, 363
 - Marking ribbons, 361
 - Masticatory muscle influence, 271
 - Mentalis m. hypertonicity, 455
 - Mouth breathing, 451
 - Muscle imbalance of dental arch envelope, 347
 - Muscular imbalance, 350-355, 377
 - Treatment methods, general, 355
 - Need for professional interaction, 343-344
 - Palpation for prematurities, 361
 - Periodontal disease, 348
 - Periodontal ligament receptors, 261
 - Phonetic occlusal evaluation, 360
 - Photocclusion for contact force, 362
 - Possible influence on total body, 349
 - Posterior slide into centric occlusion, 345
 - Primary or secondary, 365
 - Remodeling adaptation, 310, 347, 349
 - Remote health problems, effects on, 233-234
 - Screening examinations for prematurities, 360-365
 - Sleeping stresses on the face, 346
 - Sucking habits, 451
 - Tongue thrust, anterior, 347
 - Tongue thrust, lateral, 348
 - Unbalanced force on temporal bone, 60
 - Vertical dimension, 356
 - Wax bite impressions, 361
 - "Whiplash"-type injuries, 113
 - Mandible
 - Anatomy, 293
 - Condylar shapes, 297
 - Condyle, 296
 - Condyle
 - Alpha position, 308
 - Angle for x-ray, 296, 300
 - Congenital malformations, 411
 - Genetic underdevelopment of, 356
 - Growth arc, 294
 - Mandibular fossa, 53, 294
 - Mandibular Kinesiograph
 - Accuracy, 324
 - Determining malocclusion, 363
 - Determining prematurities, 363
 - Graphics of prematurities, 363-365
 - Monitoring TMJ motion, 319-325
 - Myo-monitor, 324
 - Swallowing, 274
 - Weight-bearing tests, 502-503
 - Mandibular movement, 312-333
 - 24-hour use, 401
 - Abduction
 - External pterygoid m., 252
 - Internal pterygoid m., 249
 - Temporalis m., 244
 - Anthropologic study, 327
 - Axis of rotation, 309
 - Classification of movements, 312
 - Closing engram, positional dysfunction, 309
 - Condylectomy, 312
 - Effects on mandibular closing engram, 351
 - Description of movements, 244
 - Edentulous patients, mandibular closing engram, 351
 - Elevation
 - Internal pterygoid m., 249
 - Masseter m., 247
 - Mandibular movement (continued)
 - Temporalis m., 244
 - Engram, 317
 - Envelope of motion, 314-317
 - Influence on cranial primary respiration, 270-273
 - Jaw-closing muscles, engram formation, 262
 - Jaw opening
 - Digastric m., 386
 - EMG studies, 256
 - Jaw-opening reflex, 260
 - Lateral deviation, external pterygoid m. injury, 252
 - Ligament influence on, 299
 - Measurement methods, 319
 - Motion, 87
 - Muscle imbalance, etiology of malocclusion, 311
 - Neuroanatomic basis of, 261-263
 - "Opening the head," 406
 - Posselt's envelope of motion, 314-317
 - Three-dimensional tracing, 317
 - Protrusion
 - External pterygoid m., 252
 - Internal pterygoid m., 249
 - Masseter m., 247
 - Temporalis m., 245
 - Retrusion
 - Masseter m., 247
 - Temporalis m., 245-246
 - Summary, 254
 - Translation, 327
 - Vertical velocity measurement, 323
 - With cranial primary respiration, 270
 - Mandibular positions
 - Anterior-inferior condylar position, 308
 - In relation to TMJ function, 301-309
 - Opening, lateral shift, 311
 - Physiologic centric relation, 308
 - Posterior superior centric relation, 306
 - Rest position, 302
 - Superior centric relation, 307
 - Mandibular sling, 250
 - Centric relation, 308
 - Mandibular-malleolar ligament, 300
 - Manipulation, structural
 - Iatrogenic considerations, 288
 - Iatrogenic malocclusion, 358
 - Mastication
 - Aid in diagnosing TMJ dysfunction, 434
 - Buccinator m., 251
 - Condylectomy, 309
 - Cranial motion enhancement, 271
 - Hard vs. soft foods, EMG studies, 326
 - Hyoid m. activity, 394
 - Jaw-opening reflex, 260
 - Muscle reactivity, 251
 - Masticatory muscles (see muscles, masticatory)
 - Mastoid notch, 31, 33
 - Mastoid portion and process, 30
 - Maxillary arch
 - Bite planes, 19
 - Width change, 18
 - Maxillary bone
 - Articulations, 67
 - General landmarks, 65-67
 - Motion, 67, 84
 - Processes, 65-67
 - Mechanoreceptors, 282
 - Meckel's cave, 119
 - Memory Wafer, 362
 - Meninges, 74-76

Index

- Meninges (continued)
 - Continuity with fascia, 75
 - Diaphragma sellae, 75
 - Dura mater, 74
 - Effects on cranial motion, 75
 - Endosteal layer, 74
 - Epineurium, 74
 - Falx cerebelli, 74
 - Falx cerebri, 70, 74
 - Frontal bone attachment, 61
 - Influence on cerebrospinal fluid, 75
 - Meningeal layer, 74
 - "Poles of attachment," 75
 - Reciprocal tension membrane, 75
 - Sutherland fulcrum, 75
 - Tentorial incisura, 74
 - Tentorium cerebelli, 74
 - Mental health, 513
 - Mental receptors, 283
 - Mental/emotional stress
 - Bracing, 254
 - Cranial fault relation, 221
 - Etiology of periodontal disease, 348
 - Iatrogenic considerations, 288
 - Reduced cranial motion, 513
 - Stomatognathic system relation, 239
 - TMJ dysfunction, 239-240
 - Vertical dimension loss, 254
 - Meridian (see specific muscle)
 - Muscles of mastication, 421
 - Mesial surface of tooth, 228
 - MKG (see Mandibular Kinesiograph)
 - Modular organization of body, 495-509
 - PRYT technique, 496-499
 - "Mother's delight," 467
 - Muscle couples, hyoid m's, 394
 - Muscle envelope of teeth, 442
 - Muscle strength, general, effect of mandibular posturing, 236
 - Muscle stretch reaction, 484
 - Active vs. passive, 417
 - Antagonist muscle consideration, 487
 - Fascial release technique, 420, 486
 - General description, 355
 - Spray and stretch technique, 416
 - Sternocleidomastoid m., 484
 - Trigger points, 486
 - Upper trapezius m., 485
 - Muscles
 - Dental arch orofacial envelope, forces on dentition, 442-446
 - Head-to-shoulder girdle, 275-282
 - Influence on occlusion, 350-355
 - Influence on the cranium, 150, 155
 - Influencing stomatognathic system, 89
 - Muscles, cranial
 - Epicranius (see occipitofrontalis m.), 91
 - Etiology of cranial faults, 27
 - Occipitofrontalis m., 91
 - Body language of imbalance, 147
 - Temporoparietalis m., 92
 - Muscles, ear
 - Auricularis m.
 - Anterior, 93
 - Posterior, 93
 - Superior, 93
 - Extrinsic muscles of, 93
 - Tensor tympani m., 512
 - Muscles, eye, 94-95, 111-115
 - Ciliaris m., 111, 115
 - Corrugator supercilii m., 95
 - Inferior oblique m., 111
 - Inferior rectus m., 111
 - Muscles, eye (continued)
 - Lateral rectus m., 113
 - Levator palpebrae superioris m., 95, 111
 - Medial rectus m., 111
 - Orbicularis oculi m., 94
 - Paralysis
 - Abducent n. (VI), 115
 - Oculomotor n. (III), 115
 - Trochlear n. (IV), 115
 - Sphincter pupillae m., 111, 115
 - Superior oblique m., 112
 - Superior rectus m., 111
 - Muscles, facial, electrodiagnosis, 122
 - Muscles, fast fibers, pantothenic acid in TMJ movement, 423
 - Muscles, general, etiology of cranial faults, 220
 - Muscles, hyoid, 386-399
 - Bilateral body organization, 394
 - Body language of imbalance, 395
 - Challenge, 395-399
 - Digastric m., 386-387
 - Anterior and posterior bellies, independent activity, 386
 - EMG studies, 386
 - Extirpation, 256
 - Jaw opening, 386
 - Neuromuscular spindle cell, 416
 - Palpation, 387
 - Electromagnetic field, 395
 - Examination and treatment, 395-399
 - Finding hidden dysfunction, 396
 - Geniohyoid m., 389
 - Gyroscopic feedback system, 278, 394
 - Illustrations, 392-393
 - In closed kinematic chain, 278, 281
 - Infrahyoid group, 390-392
 - Jaw-opening dysfunction, 428
 - Ligament interlink technique, 394
 - Mastication, 394
 - Mylohyoid m., 388
 - Omohyoid m., 391
 - Palpation, 392
 - Reactive muscle considerations, 395
 - Sequential correction of, 488
 - Sternohyoid m., 390
 - Sternothyroid m., 390
 - Stylohyoid m., 388
 - Suprahyoid group, 386-389
 - Synergistic activities, 383
 - Thyrohyoid m., 391
 - "Whiplash," 515
- Muscles, masticatory
 - Anatomy, 244-254
 - Buccinator m., 101, 251
 - Testing of, 101, 251
 - Deep tendon reflex, 120
 - Etiology of cranial faults, 220
 - Evaluation by isometric contraction, 425
 - Examination, 424-434
 - Examination for hypertonicity, 432
 - External pterygoid m., 252-254
 - External palpation, 427
 - Hypertonic, 300
 - Intra-oral contact, 426
 - Neuromuscular spindle cell, 416
 - Palpation of, 253, 408
 - Reflexes and meridian, 252
 - Studies indicating two muscles, 252
 - TMJ disc dysfunction, 334
 - Trigger point pain pattern, 409, 418
 - Imbalance, etiology of TMJ dysfunction, 311
 - In closed kinematic chain, 281, 278
 - Internal pterygoid m., 248-250
- Muscles, masticatory (continued)
 - Palpation of, 250, 408
 - Reflexes and meridian, 249
 - Role in TMJ dislocation, 338
 - Trigger point pain pattern, 408, 418
- Jaw closing, engram formation, 262
- Lateral pterygoid m. (see external pterygoid)
- Masseter m., 247-248
 - Jaw-jerk reflex silent period, 237
 - Measurement of belly mass, 452
 - Palpation of, 408
 - Reflexes and meridian, 248
 - Reversed origin/insertion, 248
 - Role in TMJ dislocation, 338
 - Strengthening exercises, 469
 - Trigger point pain pattern, 408, 418
- Medial pterygoid m. (see internal pterygoid)
- Myalgia, referred pain to TMJ, 413
- Palpation for dysfunction, 407-409
- Posterior capsulitis, 328
- Reactive muscle considerations, 425
- Resting activity, 254
- Synergism needed, 243
- Temporal pattern disturbance, 416
- Temporalis m., 244-246
 - Bracing, 246
 - Effect on rest position, 301
 - Fascial release technique, 246
 - Hypertonic posterior fibers, 329, 333-334
 - Hypertonicity, 433
 - Palpation of, 407
 - Posterior fibers, 327
 - Posterior fibers, hypertonic, 408
 - Reflexes and meridian, 246
 - Role in TMJ dislocation, 338
 - Squamosal sutural fault involvement, 246
 - Trigger point pain pattern, 407-408, 419
- TMJ and muscle evaluation and treatment, 401-435
- Muscles, mouth, 98-102
 - Body language
 - Hypertonic mentalis m., 445
 - Initial clues of dysfunction, 452
 - Orbicularis oris weakness, 453-454
 - Buccinator m., 101
 - Hypercontraction in swallowing, 455
 - Depressor anguli oris m., 101
 - Depressor labii inferioris m., 100
 - Etiology of imbalance, 447
 - Flaring of lips, 453-454
 - Imbalance
 - Bottle-feeding, 448
 - Cranial faults, 451
 - Deviant swallowing patterns, 447
 - Examination for, 452-454
 - Habit patterns, 448
 - Lip characteristics, 454
 - Mouth breathing, 450
 - Sleeping habits as etiology, 451
 - Sucking habits as etiology, 451
 - Increasing mobility, 463
 - Levator anguli oris m., 98
 - Levator labii superioris alaeque nasi m., 98
 - Levator labii superioris m., 98
 - Lip seal strength, 452
 - Measurement of strength, 452-454
 - Mentalis m., 101
 - Hypertonicity, 455
 - Myofunctional therapy exercises, 460-470
 - Myoscanner force transducer, 452
 - Orbicularis oris m., 102
 - AK treatment, 462, 463
 - Hypertonicity body language, 147

- Muscles, mouth (continued)
 Mouth breathing, weakness in, 451
 Normal strength, 453, 469
 Open mouth posture, 453-454
 Strengthening exercises, 461-465
 Test, 102
 Risorius m., 100
 Tensor palati m., 512
 Etiology of auditory disturbances, 411
 Tensor tympani m., etiology of auditory disturbances, 412
 Tongue, 257-258
 Zygomaticus major m., 100
 Zygomaticus minor m., 99
- Muscles, nose, 96-97
 Compressor naris m., 97
 Corrugator supercilii m.
 Glabella cranial fault, 172
 Internal frontal cranial fault, 182
 Depressor septi m., 97
 Dilator naris m., 97
 Procerus m., 96
 Glabella cranial fault, 172
 Internal frontal cranial fault, 182
 Nasal congestion, 96
- Muscles, orofacial, as a dental envelope, 442
- Muscles, postural, 275-277
 Abdominal m.
 Innominate bone motion correlation, 204
 Pelvic fault, category I, 191, 212-213
 Pelvic respiratory activity, 9
 Sagittal suture fault, 190, 191
 Coccygeus m., coccygeal fault, 210
 Head leveling, 275-277
 In closed stomatognathic system kinematic chain, 275-277
 Levator ani m., coccygeal fault, 210
 Neck extensors, action in gait, 507
 Neck flexors, medial
 External frontal fault etiology, 185
 Internal frontal fault etiology, 182
 Parietal descent correlation, 178
 Pectoralis major m. (clavicular division) bilateral weakness, 175
 Piriformis m.
 Sacral inspiration assist fault, 206, 208
 Psoas m.
 Sacral inspiration assist fault, 206, 208
 Scalene group, unilateral weakness, parietal descent correlation, 178
 Sternocleidomastoid m.
 Action in gait, 506
 External frontal fault etiology, 185
 Influence on cranium, 211
 Innervation, 133
 Internal frontal fault etiology, 182
 Muscle stretch reaction, 484
 Stress receptor, 507
 Trigger points, 419
 Universal fault, 188
 Trapezius m., innervation, 133
 Trapezius m., lower, bilateral weakness masking temporal bulge, 175
 Trapezius m., upper
 Action in gait, 506
 Influence on cranium, 211
 Muscle stretch reaction, 485
 Stress receptor, 507
 Trigger points, 419
 Universal fault, 188
- Muscles, reactive
 Buccinator m. - masseter m., 102, 251
 Hyoid m. interaction, 395
- Muscles, reactive (continued)
 In TMJ dysfunction, 415
 With the stomatognathic system, 490
- Muscles, skull, 89-103
- Muscles, slow fibers, chelated iron in TMJ movement, 423
- Muscles, tongue
 Chondroglossus m., 258
 Extrinsic m., 257
 Genioglossus m., 257
 Hyoglossus m., 258
 Inferior longitudinal m., 258
 Intrinsic m., 258
 Palatoglossus m., 258
 Styloglossus m., 258
 Superior longitudinal m., 258
 Transverse m., 258
 Vertical m., 258
- Muscular imbalance
 Cranial faults, 5
 Effects on the cranium, 89
- Myalgia, masticatory muscles, referred pain to TMJ, 413
- Myo-monitor, 378
 Used with MKG, 324
- Myocentric occlusion, 308, 378
- Myofunctional therapy, 439-470
 Age and success rate, 446
 Applied kinesiology role, 461
 Examination prior to exercises, 461
 Exercises, 460-470
 Habit pattern correction, 458-460
 Habits, detrimental, 451
 History, 439
 Introduction, 439-441
 Lateral tongue thrust, 445
 Lip character, 454
 Lip exercises, 461-465
 Lip massage, 464
 Lip mobility, increasing, 463-465
 Marshmallow twist, 462
 Masseter m. exercise, 469
 "Mother's delight," 467
 Mouth breathing, 450
 Myoscanner, 452
 Orbicularis oris m. exercises, 461-465
 Orbicularis oris m. weakness, 453
 Open mouth posture, 453-454
 Orofacial muscle imbalance, examination for, 452-457
 Orofacial muscular envelope, 442
 Permanent habit training, 470
 Sleeping habits, 451
 Strengthening anterior tongue, 469
 Sucking habits, 451
 Swallowing training, 469
 Thumb- or finger-sucking, 451
 Tongue thrust (see also under tongue thrust), 457
 Etiology, 447-452
 Studies in children, 447
 Tongue training, 465-470
 Tug-of-war exercises, 462
- Myoscanner, myofunctional therapy, 452-454
- Myositis, 413
- Myotatic reflex in muscle stretch reaction, 417
- N —
- Nares imbalance, body language, 146
- Nasal bone, 72
- Nasal concha, 72-73
- Nasal concha (continued)
 Articulations, 73
 Motion, 73
 Nasal congestion, 95, 96
 Nasal septum, 70
 Nasalis m., alar part (see dilator naris m.)
 Nasalis m., transverse part (see compressor naris m.)
 Nasolabial fold, body language, 147
 Nasolacrimal duct, 73
 Neck pain, stomatognathic dysfunction, 232
 Necrosis, avascular, 410
 Neoplasm, TMJ, 411
 Nerve entrapment (see cranial nerves)
 Nerve receptors
 Classification of, 282, 516
 Related to neurologic disorganization, 516
 Nerves, cranial (see also cranial nerves), 105-138
- Neuralgia
 Referred pain to TMJ
 Sphenopalatine ganglion, 413
 Trigeminal, 413
 Vidian, 413
- Neuralgia, glossopharyngeal, referred pain to TMJ, 413
- Neurologic disorganization, 515-523
 Body language of cranial faults, 140
 Body language of stomatognathic dysfunction, 481
 Caused by cranial faults, 170
 Determining if chemical, 519
 Determining if mental, 519
 Determining if structural, 518
 Electron poisoning, 519
 Finding hidden positive K27, 519-523
 Hyoid muscles, 395
 Idiopathic scoliosis, 23
 Inspiration/expiration assist cranial faults, 170
 Mental/emotional involvement, 240
 Model of etiology, 283-286
 Routine unswitching contraindicated, 517
 Stomatognathic system influence, 282-286
 Stomatognathic system model, 285-286
 TL to K27, 516
- Neurologic organization, stomatognathic system, effects on, 6
- Neurologic tooth
 Differential examination, 422
 Examination and treatment, 265-267
 Interaction with malocclusion, 366
 Nutritional supplementation, 267
 Periodontal ligament correlation, 265
 Recurrence, 266
 Two-hand therapy localization, 267
- Neurolymphatic reflex (see also specific muscle), 246
- Neuromuscular spindle cell
 External pterygoid m., 253
 In hyoid muscles, 396
 Interaction with malocclusion, 352
 Presence in digastric m., 416
 Presence in external pterygoid m., 416
- Neurovascular reflex (see specific muscle)
- Night guards (see bite planes)
- Nociceptors, 283
 Periodontal ligament, 260
- Nodose ganglion, 130
- Non-lever action of mandible, 325-327
- Nuchal lines, 31
- Nursing (see breast- and bottle-feeding)
- Nutrition, milk digestion by infant, 449-450
- Nutrition testing, 522

Index

Nystagmus, 127

— O —

Occipital bone
 Articulations, 50-53
 General landmarks, 50-53
 Motion, 35, 37, 52
 Ossification centers, 52
 Occiput, Lovett brother, 203
 Occlusion (see also equilibration and malocclusion), 343-378
 Acceptability of anterior slide, 308
 Angle classifications, 230-232
 Body balance, 3
 Centric, examination, 422
 Centric, physiologic centric relation, 309
 Changing of, 18
 Closed bite, 229
 Cranial faults influence on, 270
 Cranial faults, etiology of malocclusion, 311
 Crossbite, 229
 Detrimental habits, 451
 Effects of physical stress to the face, 346
 Effects of weight-bearing dysfunction, 502
 Equilibration, 375-378
 Form determining function, 310
 Gnathologic concept, 317
 Iatrogenic considerations, 287
 Evaluation of, 18
 Forces required for tooth remodeling, 443-445
 Freeway space, 229
 Increased vertical dimension interference, 357
 Head/neck ache pain patterns, 233
 Influences of muscular imbalances, 350-355
 Inlay/onlay experimental remodeling, 444
 Interplay with other functions, 349
 Introduction, 343
 Long centric, Mandibular Kinesiograph graph, 363
 Malocclusion (see also malocclusion), 345-346
 Mental/emotional effects, 239-240
 Neurologic tooth, 266
 Occlusogram, 18
 Open bite, 229
 Lateral tongue thrust, 445
 Optimum, description, 344
 Orthodontic nomenclature, 230-231
 Overbite, 229
 Overjet, 229
 Periodontal disease, 348
 Nutritional deficiencies, 239
 Phonetic evaluation, 18, 360
 Photocclusion, 362
 Physiologic centric relation, 345
 Temporalis m., posterior fibers, 244
 Physiologic rest position, 229
 Remodeling of tooth position, 347, 349
 Screening examinations for prematurities, 360-365
 Short leg, influence of, 235
 Stethoscope evaluation, 18
 Structural balance, 5
 Sucking habit considerations, 451
 TMJ disc dysfunction, 329, 334
 Vertical dimension, 305, 356
 Occlusogram, sound, 18, 360
 Occupational stress, etiology of cranial faults, 221
 Ocular lock, 23
 Cranial nerve involvement, 114-115, 170

Ocular lock (continued)
 Examination procedures (see Volume I, Chapter 9)
 Inspiration/expiration assist cranial faults, 170
 vs. eyes into distortion, 501
 Ocular lock switching caused by cranial faults, 170
 Oculo-basic technique in PRYT technique, 498
 Oculomotor n. (III), 111
 Olfactory n. (I), 70, 108
 Omnivac, fabrication of bite plane, 369
 Open bite, lateral tongue thrust, 445
 Open-mouth posture, 453
 Ophthalmic telebinocular, 110
 Ophthalmoscope examination, 110
 Optic n. (II), 109-110
 Oral diaphragm, 389
 Orbitale, 482
 Orthodontic nomenclature, 230-231
 Orthodontic treatment, need for, 345
 Orthophoria, 114
 Osteoarthritis
 Radiologic analysis, 410
 TMJ, 328, 410
 Osteochondritis, TMJ, 410
 Osteoma, condylar head, 411
 Otomandibular syndrome, 234
 Overbite
 Definition, 229
 Thumb-sucking, 451
 Overjet, definition, 229

— P —

Pain
 Along suture, 147
 Junction of 1st rib, clavicle, and sternum, 211
 Referred, 514
 Palatal rugae, 456
 Palatine bone
 Articulations, 67-69
 General landmarks, 67-69
 Motion, 69, 86
 Processes, 67
 Pantothenic acid, fast muscle fibers in TMJ movement, 423
 Papillae, tongue, 256
 Parietal bone
 Articulations, 65
 General landmarks, 64-65
 Motion, 65, 83
 Parietal descent cranial fault (see also cranial faults, parietal descent), 177
 Parotid gland
 Glossopharyngeal n. (IX), 128
 Innervation, 118
 Patient consultation, body language of cranial faults, 143-144
 Patient scheduling for treatments, 489
 Payne technique, 457
 Pelvic faults
 Category I, 211-213
 Block adjusting technique, 211
 Challenge, 211
 Etiology of cranial faults, 211
 Examination, 211-212
 Leg length, 211
 Muscle involvement, 212
 Pain pattern, 211
 Shoulder girdle adaptation, 211
 Therapy localization, 211

Pelvic faults (continued)
 With cranial faults, 170
 Category II, 211
 Influence on stomatognathic system, 239
 TMJ dysfunction, 239
 Cloacal synchronization correlation, 214-216
 Coccyx, 210
 Challenge, 210
 Conditions correlating, 210
 Correction, 210
 Etiology, 210
 Muscle correlation, 210
 Pain location, 210
 Therapy localization, 210
 Piriformis m. correlation, 206
 Psoas major m. correlation, 206
 Recurrent, 220-222
 Sacral expiration assist, 207-209
 Challenge, 208
 Conditions correlating, 209
 Correction, 209
 Mechanics of correction, 207
 Mechanics of fault, 207
 Muscle correlation, 208
 Pain location, 208
 Therapy localization, 207
 Sacral inspiration/expiration assist, bilateral involvement, 209
 Sacral inspiration assist, 205-207
 Challenge, 206
 Conditions correlating, 206
 Correction, 206
 Mechanics of correction, 206
 Mechanics of fault, 205
 Muscle correlation, 206
 Pain location, 206
 Therapy localization, 206
 Three-dimensional characteristics, 206
 Sacral respiratory fault, universal fault, 189
 Sacral wobble, 217-218
 Atlanto-occipital counter torque, 218
 Challenge, 217
 Correction, 218
 Examination, 217
 Therapy localization, 217
 Walking gait, 217
 Sacral, universal cranial fault, 189
 Sacrum with glabella cranial fault, 172
 Pelvic motion, abdominal m. involvement, 9
 Pelvis (see also pelvic faults, sacral primary respiration, sacrum, and coccyx), 197-222
 Anatomy, 198-201
 Coccyx
 Anatomy, 199
 Coccygodynia, 210
 Lovett brother, 210
 Cranial-sacral motion, dura mater, 197
 Innominate bone
 Anatomy, 199-200
 Lovett brother, 204
 Introduction, 197
 Motion, 201-205
 Coccygeal, 203
 Sacrum, 201
 Sagittal plane, 201
 Sacroiliac articulation
 Anatomy, 200
 Motion, 200
 Sacrum, anatomy, 198
 Perimetry, 110
 Periodontal disease, 348
 Bite plane therapy, 374
 Blood sugar handling stress, 239

- Periodontal disease (continued)
 Effects of orofacial muscle imbalance, 446
 Nutritional deficiencies, 239
 Periodontal ligament, 259-260
 Jaw-closing muscle engram formation, 262
 Jaw-opening reflex, 260
 Anterior digastric m., 387
 Mandibular closing engram, 351
 Referred pain, 261
 Types of receptors, 260
 Periodontal transmission mechanism, 259
 Periodontitis, body language of stomatognathic dysfunction, 480
 Periodontosis, 480
 Peripheral nerve entrapment (see also cranial nerves), 105-107
 Carpal tunnel syndrome, 105
 Quantitative measurement, 106
 Peripheral vision
 Confrontation test, 110
 Optic n. (II) dysfunction, 110
 Pharyngeal paralysis, vagus n. (X), 132
 Phonation (see also speech)
 Bilateral brain activity, 435
 In diagnosing TMJ dysfunction, 435
 Infrahyoid m., 390-392
 Vagus n. (X) dysfunction, 132
 Phonetic occlusal evaluation, 360
 Phonetics, establish vertical dimension, 372
 Photoclusion, 362
 Physiologic centric relation (see centric relation, physiologic)
 Physiologic motion, cranial faults, 149
 Physiologic rest position (see rest position)
 Picard's mouth, 449
 Pineal gland, 523
 Cranial dysfunction, 22
 Pitch-Roll-Yaw-Tilt (PRYT)
 Effect on stomatognathic examination, 435
 Effects on cranial mechanism, 90
 Etiology of neurologic disorganization, 521
 Pitch, 497
 Roll, 498
 Technique, 496-499
 Tilt, 499
 Yaw, 498
 Pituitary gland dysfunction, 514
 Cranial etiology, 22
 Polyarteritis nodosa, referred pain to TMJ, 413
 Pons, intrapontine lesion, 124
 Porion, 482
 Posselt's envelope of motion, 314
 Engram of habitual arc, 309
 Postural analysis
 Head leveling, 482
 Modular organization, 495
 Plumb-line analysis, 496
 Shoulder girdle, 483
 Stomatognathic system, 481-484
 Pregnancy, motion in sacroiliac, 200
 Prematurity
 Definition, 229
 Periodontal ligament receptors, 261
 Primary respiratory motion, explanation of, 9
 Proprioceptors
 Equilibrium, 6
 Mandibular movement integration, 261-263
 Periodontal ligament, 260
 Proprioceptors, equilibrium, 282
 Eyes into distortion (EID), 500
 Grouped by location, 500
 Influence on body organization, 483
 Involvement with stomatognathic system, 235
 Proprioceptors, equilibrium (continued)
 Stomatognathic system, 3
 Vertigo, 512
 Proprioceptors, muscle
 Stomatognathic postural muscles, 484
 Treatment in TMJ dysfunction, 415
 Proximal surface of tooth, 228
 PRYT technique (see Pitch-Roll-Yaw-Tilt)
 Psychological stress, relation to cranial faults, 221
 Psychophysiologic (see also mental/emotional stress)
 Bruxism, 374
 Stomatognathic system dysfunction, 352
 Psychophysiologic theory
 Definition, 232
 Etiology of myofascial pain-dysfunction, 240
 Pterion, 30
 Pterygoid hamulus, 45
 Pterygoid plates, articulation with pyramidal process, 86
 Pterygoid processes, 45
 Pubis, anatomy (see also innominate bone), 199
 Pyramidal process, 68
 Motion, 86
- Q —
- Quadratus bone, 327
- R —
- Radiologic analysis (see x-ray)
 Reactive muscle (see muscles, reactive)
 Receptor-tonus technique, TMJ, 420
 Receptors, nerve, classification of, 282
 Recidivism, 3, 5
 Cranial faults, 220-222
 From weight-bearing problems, 502
 Hidden problems found with EID, 500-502
 Pelvic faults, 220-222
 Reciprocal tension membrane, 75
 Referred pain, periodontal ligament receptors, 261
 Reflex arc, 282
 Reflex, gag, 128
 Reflex, jaw, 120
 Masseter m. silent period, 237, 416
 Masticatory muscles, 120
 Reflex, jaw-opening
 Anterior digastric m., 387
 Periodontal ligament receptors, 261
 Reflex, myotatic, 417
 Reflexes, equilibrium, 6, 23
 Acoustic n. (VIII), 126
 Babinski-Weil test, 127
 Cloacal synchronization technique, 214-215
 Closed kinematic chain, 280
 Postural muscles, 275
 Universal cranial fault correlation, 188
 Reflexes, neurolymphatic (see specific muscle)
 Muscles of mastication, 421
 Reflexes, neurovascular (see specific muscle)
 Muscles of mastication, 421
 Remodeling
 Bone, 345
 Teeth in malocclusion, 347, 349
 Example of, 363-364
 Respiration, pelvic motion parallel, 9
 Respiration, thoracic, correlation with
 Cranial motion, 139-141
 Respiration, thoracic (continued)
 Cranial motion parallel, 9, 18
 Pelvic motion, 139
 Sacral motion, 139
 Sphenoid and occipital bone movement, 140
 Rest position, 302
 Determination of, 302
 Factors which influence, 305
 Malocclusion, effect on, 303
 Mandibular Kinesiograph, 302-304
 Measurement by speaking "Boston," 304
 Measurement methods, 302-305
 Monitoring with Mandibular Kinesiograph, 322
 Myo-monitor, 302
 Physiologic definition, 229
 Postural influence on, 302
 Registering with Myo-monitor, 378
 Transcutaneous electrical neural stimulation (TENS), 302
 Vertical position, 356
 Retrograde lymphatic, treatment for bruxism, 375
 Rhinitis, 108, 512
 External frontal cranial fault, 185
 Internal frontal cranial fault, 182
 Rinne's test, 126
 "Rock shaft and pivot," 57, 79
 Roentgenographic analysis (see x-ray)
 Roling, 420
 Roll, PRYT technique, 498
- S —
- Sacral expiration assist (see also pelvic faults), 207
 Sacral inspiration assist (see also pelvic faults), 205
 Sacral motion
 Axis of rotation, 201
 Cranial motion correlation, 18, 201-203
 Dural mechanism, 9, 197
 Spinal motion correlation, 201-203
 Sacral primary respiration, 139
 Sacral wobble (see also pelvic faults, sacral wobble), 217-218
 Sacroiliac, motion, 200-201
 Sacrum (see also pelvic faults, sacrum)
 Anatomy, 198
 Articulation with innominate bone, 200
 Base, 198
 Lovett brother, 203, 210
 Sagittal suture cranial fault (see also cranial faults, sagittal suture), 190-191
 Salivary glands
 Diagnosis, 413
 Hypersecretions/hyposecretions, vagus n. (X) dysfunction, 132
 Schizophrenia, 521
 Scoliosis, body language of stomatognathic dysfunction, 481
 Shiatsu therapy, 418
 "Shifty-eyed look," 114
 Shoulder, frozen, spinal accessory n. (XI) involvement, 134
 Shoulder/arm syndrome
 Body language of stomatognathic dysfunction, 481
 Examination to find cause, 491
 Sinus
 Occipital, 74
 Paranasal, 513

Index

- Sinus (continued)
 - Sagittal, 74
 - Transverse, 74
- Sinusitis
 - Body language of stomatognathic dysfunction, 481
 - Etiology of referred pain, 412
 - External frontal cranial fault, 185
 - Internal frontal cranial fault, 182
 - Treatment comparison of specialties, 404-406
 - Vidian neuralgia, 413
- Skull asymmetry, 143-144
- Skull motion (see cranial motion)
- Skull muscles (see muscles), 89-103
- Sleeping position
 - Advantages of back sleeping, 346
 - Etiology of malocclusion, 346
 - Orofacial muscle imbalance, 451
- Small intestine, lymphatic congestion, 95-96
- Snoring, vagus n. (X) dysfunction, 132
- Speaking space, closest, 304, 373
- Speech (see also phonation)
 - Influence on stomatognathic system, 352
 - Related to hearing deficit, 512
- Speech irregularities, body language of stomatognathic dysfunction, 479-480
- Sphenobasilar expiration assist cranial fault (see also cranial faults), 167
- Sphenobasilar extension
 - Definition, 35
 - Sacral motion correlation, 201-203
 - Sphenoid and temporal bone motion, 60
 - Spinal motion correlation, 201-203
- Sphenobasilar flexion
 - Definition, 35
 - Ethmoid bone motion, 71
 - Frontal bone motion, 61
 - Maxillary bone motion, 67
 - Occipital bone motion, 52
 - Palatine bone motion, 69
 - Parietal bone motion, 65
 - Sacral motion correlation, 201-203
 - Sphenoid bone, 48
 - Spinal motion correlation, 201-203
 - Temporal bone motion, 56, 57
 - Vomer bone motion, 71
- Sphenobasilar inspiration assist cranial fault (see also cranial faults), 165
- Sphenobasilar junction, 34
 - Cancellous bone, 13
 - Motion, 10, 35, 37, 48
 - Ossification of, 13
- Sphenoid bone
 - Articulations, 43-49
 - Axis of rotation, 48, 79
 - Cranial motion, key bone in, 43
 - General landmarks, 43-49
 - Lovett brother, 203, 210
 - Motion, 35, 37, 48
 - Pterygoid process, 45
 - Sutures, 48
- Sphenosquamous pivot, 49, 79, 81-82
 - Location, 48
 - Sphenoid and temporal bone motion, 59
 - Temporal bone, 57
- Spinal accessory n. (XI), 132-134
- Spinal balance, stomatognathic system, 3-5
- Spinal dysfunction, influence on stomatognathic system, 435
- Spinal motion, 201-203
 - Cranial-sacral motion correlation, 201-203
- Splint (see also bite planes), 365-375
 - Protection of teeth, 374
- Spray and stretch technique
 - In TMJ dysfunction, 416
 - Temporalis m., 246
- Squamosal suture cranial fault (see also cranial faults, squamosal suture), 191-192
- SS pivot (see sphenosquamous pivot)
- Stereopsis, 110
- Stethoscope, occlusion evaluation, 18
- Stomatognathic area
 - Body language of dysfunction, 480
 - Cranial interdependence, 269-289
 - Definition, 225
 - Homuncular representation, 263-265
 - Introduction, 225
 - Mandibular influence on cranial function, 270-273
 - Orofacial muscle imbalance, 442-457
 - Orthodontic nomenclature, 230-231
 - Sequential order for correction of, 488
 - Terminology, 225-231
- Stomatognathic system
 - Benefits of AK evaluation, 237
 - Body language of dysfunction, 478-481
 - Body organization, effects on, 6
 - Controversy among practitioners, 236
 - Cranial nerve dysfunction, general discussion, 355
 - Cranial-stomatognathic area interdependence, 269-289
 - Definition, 3
 - Documenting examination findings, 478
 - Dysfunction
 - Auto accident example, 402
 - Treatment comparison of specialties, 402-406
 - Endocrine disorders, 514
 - Equilibration correlation, 375
 - Etiology of
 - Cranial faults, 144
 - Neurologic disorganization, 282-286, 522
 - Examination as an integrated system, 477-493
 - Form vs. function, 310
 - Frankfurt plane, 482
 - General symptoms of involvement, 481
 - Head-to-shoulder girdle musculature, 275-282
 - Hyoid muscle balance, 278-280, 394
 - Iatrogenic considerations, 286-289
 - Intersystem reactivity, 490
 - Introduction, 3, 225-240
 - Mandibular opening reflex, 260
 - Mandibular sling, 250
 - Muscle proprioceptive dysfunction, 352-354
 - Muscle treatment methods, 355
 - Neurologic disorganization, 515-523
 - Neurologic disorganization model, 285-286
 - Normal jaw aperture, 255
 - Occlusion, 343-378
 - Organization with total body function, 495-509
 - Pain symptoms, common, 232
 - Patient scheduling for treatment, 489
 - Periodontal ligament receptors, 261
 - Periodontal transmission mechanism, 259
 - Postural analysis, 481, 482-483
 - Postural muscles, 275-277
 - Recidivism, 489
 - Eyes into distortion (EID), 500-502
 - Gait testing, 505-509
 - Habit patterns, 491
 - Occupational considerations, 491
 - PRYT technique, 496-499
 - Weight-bearing tests, 502-505
 - Relation with various health problems, 511-
- Stomatognathic system (continued)
 - 524
 - Remote symptoms, 233
 - Role in general health problems, 232-238
 - Sequential steps in examination and correction, 482-488
 - Spinal column, integration with, 3
 - Structural balance, 3
 - Tinnitus, incidence with, 233
 - TMJ evaluation, 401-435
 - Total body, integration with, 3
 - Trauma, 481
 - Triad of health, 238-240
- Stress receptor (see also specific muscle)
 - Muscles of mastication, 421
 - Sternocleidomastoid m., 507
 - Upper trapezius m., 507
- Structural imbalance, etiology of cranial faults, 26
- Structure, relation to stomatognathic system, 238
- Subluxation, TMJ, 411
- Subluxation, occipital, hyoid m. involvement, 394
- Subluxation, upper cervical
 - Cause of neurologic disorganization, 286
 - Persistent, 188
- Subluxations, spinal, influence on stomatognathic system, 355
- Sucking habits, orofacial muscle imbalance, 451
- Surgery, stomatognathic, need for, 345
- Sutherland fulcrum, 75
- Sutural bevel, internal and external, 41
- Sutural bones (see wormian bones)
- Suture, nerve fibers, 13
- Suture development, dependent on muscle balance, 16
- Suture types
 - Amphiarthrodial, 42
 - Dentate, 41
 - False, 41
 - Gomphosis, 42
 - Limbus, 41
 - Plane, 41
 - Schindylesis, 42
 - Serrate, 41
 - Squamous, 41
 - Symphysis, 42
 - Synchondrosis, 42
 - True, 41
- Sutures
 - Coronal, 61, 65
 - Cruciate, 33
 - Experiment on development, 16
 - Fixation experiment, 15
 - Frontoethmoid, 71
 - Frontomaxillary, 67
 - Frontonasal, 72
 - Frontosphenoidal, 49, 61
 - Frontozygomatic, 70
 - Histology, 13, 40
 - Intermaxillary, 67
 - Internasal, 72
 - Lambdoidal, 53, 65, 83
 - Metopic, 16, 61, 62, 180
 - Metopic remnant, 32
 - Nasomaxillary, 67, 72
 - Nerve receptors in, 20
 - Occipitomastoid, 53, 57, 58
 - Early ossification, 16
 - Palatomaxillary, 67
 - Parietomastoid, 57, 65
 - Pathologic ossification, 16

- Sutures (continued)
 Premature ossification, 15-16
 Sagittal, 65
 Sphenothmoid, 81
 Sphenoparietal, 49, 65
 Sphenopetrous, 48
 Sphenozygomatic, 49, 70
 Squamosal, 57, 65
 Syndesmology, 39-42
 Temporosphenoidal, 49, 57
 Temporozygomatic, 70
 Zygomaticomaxillary, 67, 70
- Swallowing, 273-274
 24-hour period, 401
 Cinefluorographic studies, 447
 Deviant pattern
 Bottle-feeding of infants, 448
 Cranial faults, 451
 Examination, 452-457
 Habit pattern correction, 458-460
 Influence on hard palate, 456
 Retraining of tongue, 465-470
 Dysphagia, vagus n. (X) dysfunction, 132
 Facial grimace, 454
 Force on teeth, 445
 Hyoid bone movement, 273, 274, 457
 Hyoid muscle imbalance, 435
 In diagnosing TMJ dysfunction, 435
 Influence on stomatognathic system, 352
 Intercuspal, 455
 Myofunctional therapy training, 469
 Rate per day, 273
 Tongue placement during, 273, 447
 Tongue thrust, 446
 Sweating, gustatory, 132
 Switching (see neurologic disorganization)
 Syndesmology, 39-42
 Syndrome
 "Cheerleader's," 410
 Costen's, 232
 Otomandibular, 234
 Thoracic outlet, 178
 Synkinesis, 103, 124, 479
- T —
- Tachycardia
 Glossopharyngeal neuralgia, 129
 Vagus n. (X) paralysis, 132
- Taste
 Anodal galvanic stimulation, 124, 128
 Examination for, 124
 Facial n. (VII), 124
 Anterior two-thirds of tongue, 122
 Glossopharyngeal n. (IX), 128
 Mandibular n., 118
 Tongue
 Anterior two-thirds, 122
 Posterior one-third, 128
- Tearing, examination, 124
- Tears
 Excess, 479
 Lack of, 94, 479
- Teeth (see also tooth)
 Anatomy, 226-227
 Challenge and manipulation, 265-267
 Diastemata, 455
 Equilibrium theory of tooth position, 442
 Etiology of referred pain, 412
 Forces applied, 260
 Intrusion, 254
 Reduced vertical dimension, 357
- Teeth (continued)
 Loss of tissue, 259
 Minimal forces required for movement, 347
 Pain, neurologic tooth, 266
 Pain from trigger points, 486
 Periodontal ligament, 259
 Periodontal transmission mechanism, 258
 Position remodeling, 349
 Prematurity, 229
 Stability in alveolar socket, 347
 Stabilization of, splint therapy, 371
 Surface definitions, 228
- Temporal bone
 Articular eminence, 53, 54
 Articulations, 57-60
 As a "connecting bone," 60
 Associated muscles, 60
 Axis of rotation, 79, 80
 Forces applied by mastication, 60
 General landmarks, 53-60
 Lovett brother, 204
 Mandibular fossa, 53, 54, 295
 Mastoid portion, 53, 54
 Mastoid process, 53-55
 Motion, 10, 36, 37, 49, 56, 57
 Petrous portion, 54
 "Wobbling wheel" effect, 80
 Temporal bulge cranial fault (see also cranial faults, temporal bulge) 174
 Temporal tap, temporalis m., hypertonic posterior fibers, 244, 334
 Tentorial incisura, 74
 Tentorium cerebelli, 74
 Terminal hinge position, TMJ, 313
 Therapists, myofunctional, 441
 Therapy localization
 Cranial faults (see specific cranial fault)
 Hyoid m., 396
 Nonspecificity, 152
 TMJ, 424
 Thermoreceptors, 283
 Thoracic outlet syndrome, 178
 Thumb-sucking, 451
 Digestive system dysfunction, 458
 Habit pattern correction, 458
 Thyroid cartilage, 384
 Movement, 391
 Thyroid gland, cranial faults, 514
 Tic douloureux (see trigeminal neuralgia)
 Tics, facial, 479
 Tilt, PRYT technique, 499
 Tinnitus, 23, 479
 Cochlear n. dysfunction, 126
 Otomandibular syndrome, 234
- TMJ, 291-339
 Ankylosis of joint, 411
 Arthritis, 328
 Degenerative joint disease, 410
 Infectious, 411
 Psoriatic, 411
 Rheumatoid, 410
 Arthropathy, 307
 Articular eminence, 295
 Articulation, 294-301
 Auditory disturbances, 234
 Avascular necrosis, 410
 Basion relationship, 296
 Body language
 Mandibular position, 147
 Of dysfunction, 402, 480
 Centric occlusion
 Definition, 306
 Examination and evaluation of, 422
- TMJ (continued)
 Centric relation
 Anterior-inferior condylar position, 308
 Definition, 306
 EMG studies, 307
 Most superior position, 307
 Retruded superior position, 306-309
 Centric relation/centric occlusion, 306-309
 Cervical traction, effects of, 411
 "Cheerleader's" syndrome, 410
 Condylar congenital anomalies, 411
 Condyle position classification, 300-301
 Disc (see also disc dysfunction, TMJ), 328-333
 Anatomy, 297
 Anterior dislocation, 337
 Anterior displacement, 307
 Histologic changes with age, 297
 Normal motion, 330
 Synovial membrane, 298
 Wear factors, 298
 Dislocation, 338
 Etiology, 338
 Muscular imbalances, 338
 Recurrent, 338
 Surgery, 338
 Dysfunction, cranial faults, 23
 Embryologic development, 291
 Equilibration, 375-378
 Evaluation and treatment, 401-435
 Examination, 421-435
 Centric occlusion, 422
 Cervical fixations, 435
 Cervical subluxations, 435
 Chewing as diagnostic aid, 434
 Flow chart, 436
 Forced transverse movement, 432
 Forced transverse movement with TL, 432
 Hypertonic jaw-closing muscles, 423-424
 Integration of multiple movements, 433
 Isometric masticatory muscle contraction, 425
 Jaw-closing muscles, 429
 Jaw closing with therapy localization, 428
 Jaw forced open without therapy localization, 423
 Jaw movement without therapy localization, 423
 Jaw opening with therapy localization, 426
 Observing body language, 406
 Palpation, 406
 Palpation of masticatory muscles, 407-409
 Passive range of joint motion, 407
 Phonation, 435
 Sagittal jaw motion with therapy localization, 425
 Swallowing, 435
 Therapy localization to TMJ, 424-432
 Therapy localization without motion, 425
 Transverse jaw motion with therapy localization, 431
 "Wagging" jaw side to side, 431
 Weight bearing, 435
 Without therapy localization to TMJ, 422-424
 Extracapsular dysfunction, 412-414
 Fascial release technique, 420
 Freeway space, 305
 Increased vertical dimension interference, 357
 General description, 291
 Gouty arthritis, 410
 Iatrogenic trauma, 411

Index

TMJ (continued)

- Importance of muscle balance, 292
- In the newborn, 292
- Interincisal distance as diagnostic aid, 255
- Interincisal distance, normal, 255
- Intracapsular pathology, 410-411
- Jaw aperture, normal, 255
- Jaw motion dysfunction, cervical extension-flexion, 434
- Jaw opening
 - As body language of dysfunction, 406
 - EMG studies, 256
- Jaw opening, maximal
 - Digastric m., anterior belly, 394
 - EMG studies, 255
- Jaw-closing dysfunction, 429
 - Masseter m., 429-430
 - Temporalis m., 430
- Jaw-closing engram, postural changes, 435
- Jaw-jerk reflex, silent period, 237
 - Prolonged in dysfunction, 416
- Jaw-opening dysfunction
 - Digastric m., anterior belly, 428
 - External pterygoid m., 426
 - Reflex, periodontal ligament receptors, 261
- Joint classifications, 312
- Lever classifications, 325-327
- Ligaments, 299
- Luxation (see also TMJ dislocation), 411
- Malocclusion
 - Etiology of referred pain, 412
 - Examination procedures before equilibration, 359-375
- Mandibular fossa, 295
- Mandibular movement, 312-318
 - Neuroanatomic basis of, 261-263
- Mandibular positions, 301-309
- Mandibular sling, 250
- Mandibular-malleolar ligament, 291
- Muscle pain patterns, 233
- Muscle stretch reaction, 417
- Neoplasms, 411
- Neurologic tooth, 422
- Occlusal symptoms, 233
- Occlusion (see also occlusion, malocclusion, and equilibration), 343-378
 - Equilibration concepts, 317-318
- Osteoarthritis, 410
- Osteochondritis, 410
- Osteomas, 411
- Palpation, 319
 - For dysfunction, 406
- Pathological vs. functional conditions, differential diagnosis, 409-414
- Pathology, indicated by therapy localization, 425
- Periodontal disease, nutritional deficiencies, 139
- Physiologic centric relation, definition, 308
- Posterior capsulitis, 328
 - Palpation, 407
- Potential joint spaces, 298
- Psychophysiologic theory of dysfunction, 240
- Reactive muscles, buccinator m.-masseter m., 251
- Referred pain etiology, 412-414
 - Auditory system, 412
 - Glossopharyngeal neuralgia, 413
 - Herpes zoster, 413
 - Masticatory muscle myalgia, 413
 - Parotid gland infections, 413
 - Polyarteritis nodosa, 413

TMJ (continued)

- Salivary gland diseases, 413
 - Sinusitis, 412
 - Teeth, 412
 - Trigeminal neuralgia, 413
 - Remodeling adaptation, 310
 - Remodeling, pathological, 328
 - Retruded superior position
 - Cause of anterior disc, 329
 - Definition, 306
 - Rotation and translation, 313-318
 - Lateral movements, 316
 - Posselt's envelope of motion, 314
 - Sphenopalatine ganglion neuralgia, 413
 - Stabilization by muscles/ligaments, 299
 - Subluxation, 411
 - Syndrome, recognition of, 236
 - Trauma, effects of, 410
 - Treatment methods
 - Acupuncture meridian connectors, 420
 - Cranial-sacral faults, 414
 - Fascial release technique, 420
 - Muscle proprioceptors, 415
 - Neurologic reflexes, 420
 - Spray and stretch technique, 416
 - Trigeminal neuralgia, 121, 413
 - Vertical dimension
 - Bite plane therapy, 370-374
 - Examination for, 422
 - Vidian neuralgia, 413
 - Weight vs. non-weight bearing, 295-298
 - Mandibular fossa, 307
- ### Tongue
- Action during breast- and bottle-feeding, 449
 - Activity during swallowing, 273
 - Anatomy, 256-258
 - Ankylotic, 456
 - Impressions of teeth, 456
 - Measuring extensor strength, 452
 - Middle portion strengthening, 469
 - Motion, 257-258
 - Motor innervation, 136
 - Muscles of, 256-258
 - Myofunctional therapy
 - Anterior function, 465-467
 - Middle function, 467
 - Posterior function, 468
 - Papillae, 256
 - Position during mouth breathing, 451
 - Position during swallowing, 447
 - Strengthening anterior portion, 469
 - Taste
 - Anterior two-thirds, 122
 - Facial n. (VII), 122
 - Glossopharyngeal n. (IX), 128
 - Posterior one-third, 128
- ### Tongue extension, lateral, occipital subluxation, 394
- ### Tongue thrust
- Anterior, 347
 - Facial grimace, 454
 - Bottle- vs. breast-feeding, 448
 - Diastemata, 455
 - Etiology, 447-452
 - Incidence in children, 447
 - Lateral, 348
 - Appearance of tongue, 456
 - Open bite, 445
 - Side sleeping habit, 451
 - Malocclusion as etiology, 452
 - Masticatory muscle balance, swallowing, 446
 - Mouth breathing, 451
 - Mouth breathing etiology, 448

Tongue thrust (continued)

- Myofunctional therapy training, 465-470
- Sucking habits, 451
- Transitional thrusters, 447
- Ultraviolet light analysis, 457
- Tonsillectomy, external pterygoid m. damage, 252
- Tooth (see also teeth)
 - Eruption, mechanism of, 371
- Extraction
 - Etiology of cranial faults, 287
 - Iatrogenic malocclusion, 358
- Intrusion
 - Bracing, clenching, 254
 - Lateral tongue thrust, 445
 - Over-increased vertical dimension, 371
- Loss, orofacial muscle imbalance etiology, 446
- Mobility, malocclusion, 347
- Position
 - Determined by muscle balance, 443
 - Equilibrium theory of, 444
 - Experimental remodeling, 444
 - Force vs. time for remodeling, 444
 - Forces required for remodeling, 443-445
 - Lateral tongue thrust, 445
 - Remodeling, experimental, 444
- "Tooth theory," definition, 232
- "Tooth-muscle" theory, definition, 232
- Torticollis, chronic-clonic-tonic intermittent, gait exercises, 509
- Torus palatinus, 456
- Traction, influence on stomatognathic system, 287, 515
- Transcutaneous electrical neural stimulation (TENS), used in equilibration, 378
- Trauma, cause of cranial faults, 143
- Trauma, general, 515
- Triad of health
 - Stomatognathic system correlation, 238-240
 - Structure, malocclusion, 239
- Trigeminal n. (V)
 - Mandibular branch, 118
 - Maxillary branch, 116-118
 - Ophthalmic branch, 116
 - Parasympathetic distribution, 120
 - Sensory distribution, 120
- Trigeminal neuralgia
 - Classifications, 121
 - Dental intervention, 121
 - Etiology, 119
 - Idiopathic, 121
 - Incident rate, 121
 - Medical intervention, 121
 - Symptomatic, 121
 - Traumatic neuroma, 121
- Trigger points
 - External pterygoid m.
 - Pain patterns, 409
 - Referred pain pattern, 418
 - General description, 355
 - Hyoid muscle treatment, 396
 - Hypertonic TMJ muscles, examination, 424
 - In TMJ dysfunction, 416
 - Internal pterygoid m.
 - Pain pattern, 408
 - Referred pain pattern, 418
 - Masseter m.
 - Pain pattern, 408
 - Referred pain pattern, 418
 - Muscle stretch reaction, active vs. passive, 417
 - Palpation of in TMJ, 417-419

- Trigger points (continued)
 - Referred pain, 514
 - Referred TMJ pain, 413, 417-419
 - Sternocleidomastoid m.
 - Referred pain, 486
 - Referred pain pattern, 419
 - Sternal vs. clavicular division, 486
 - Temporalis m.
 - Pain pattern, 407-408
 - Referred pain pattern, 419
 - Treatment methods in TMJ, 417
 - Upper trapezius m.
 - Referred pain, 486
 - Referred pain pattern, 419
 - Trismus, 337
 - Trituration, 244
 - Trochlear n. (IV), 112
 - Tumor, frontal lobe, 108
- U —
- Universal cranial fault (see also cranial faults, universal), 187-189
- V —
- Vagotomy, 132
 - Vertebra, cervical, correction with glabella
 - cranial fault, 173
 - Vertebral fixations, dorsolumbar, masking
 - temporal bulge fault, 175
 - Vertical dimension
 - Bite plane therapy, 370-374
 - Bite restoring vs. bite opening, 371
 - Closest speaking space, 373
 - Definition, 356
 - Evaluation for, 422
 - Increased
 - Contraindications, 372
 - Etiology, 357
 - Temporary reduction of symptoms, 373
 - Mandible, genetic underdevelopment, 356
 - Occlusion, 356
 - Optimum approach for determination, 372
 - Optimum occlusion, 344
 - Phonetic approach, 372
 - Postural, 356
 - Reduced, 356
 - Bruxism and bracing as etiology, 254, 357
 - Etiology, 356
 - Intrusion of teeth, 357
 - Symptoms of, 356
 - TMJ disturbances, 356
 - Tooth eruption interference, 357
 - Rest position, 356
 - Vertigo, 142
 - Otolith vs. cervical, 512
 - Visual acuity
 - Body language of cranial faults, 142, 479
 - Cardinal positions of gaze, 114
 - Internal frontal cranial fault, 182
 - Optic n. (II) dysfunction, 110
 - Reading card, AMA, 110
 - Snellen eye chart, 110
 - Visual disturbances, 114-115
 - Alternate cover test, 114
 - Anaglyph test, 115
 - Body language of cranial faults, 142
 - Cover test, 114
 - Cranial faults, 23
 - Esotropia, 114
 - Visual disturbances (continued)
 - Examination to find cause, 491
 - Eye muscle paralysis, 115
 - Glaucoma, 142
 - Ocular lock, 115
 - Orthophoria, 114
 - Pupil dilation, 115
 - "Shifty-eyed look," 114
 - Visual phoria, 110
 - Visual righting reflexes, neurologic disorganization, 285
 - Vitamin B deficiency, tongue symptoms, 136
 - Vitamin C overdose, 522
 - Vitamin C deficiency, periodontal disease, 348
 - Vitamin E overdose, 522
 - Vomer bone, 71
 - Articulations, 71
 - Motion, 71, 85
- W —
- Wax bite, impressions for prematurities, 361
 - Weber's test, 126
 - Weight-bearing
 - Hyoid muscle imbalance, 396
 - Recurrent cranial-pelvic faults, 222
 - Sacral expiration assist, 208
 - Sacral inspiration assist, 206
 - Testing, 502-505
 - "Whiplash," 25
 - Abducent n. (VI) dysfunction, 113
 - Body language of stomatognathic dysfunction, 481
 - Cause of cranial faults, 143
 - Influence on stomatognathic system, 515
 - Lambdoidal suture cranial fault, 193
 - Winking, 94
 - "Wobbling wheel" effect, 56
 - Wolff's law, 346
 - Wormian bones, 25, 57
 - Cranial motion enhancement, 39
- X —
- X-ray
 - Cranial evaluation, 18
 - Study of cranial-pelvic correlation, 18
 - TMJ
 - Degenerative joint disease, 410
 - Infectious arthritis, 411
 - Neoplasm, 411
 - Osteochondritis, 410
 - Rheumatoid arthritis, 410
- Y —
- Yaw, PRYT technique, 498
- Z —
- Zinc, neurologic tooth supplementation, 267
 - Zygomatic bone
 - Articulations, 70
 - General landmarks, 69-70
 - Motion, 70, 87
 - Processes, 69-70
 - Zygomatic suture cranial fault (see also cranial faults, zygomatic suture), 194-195

